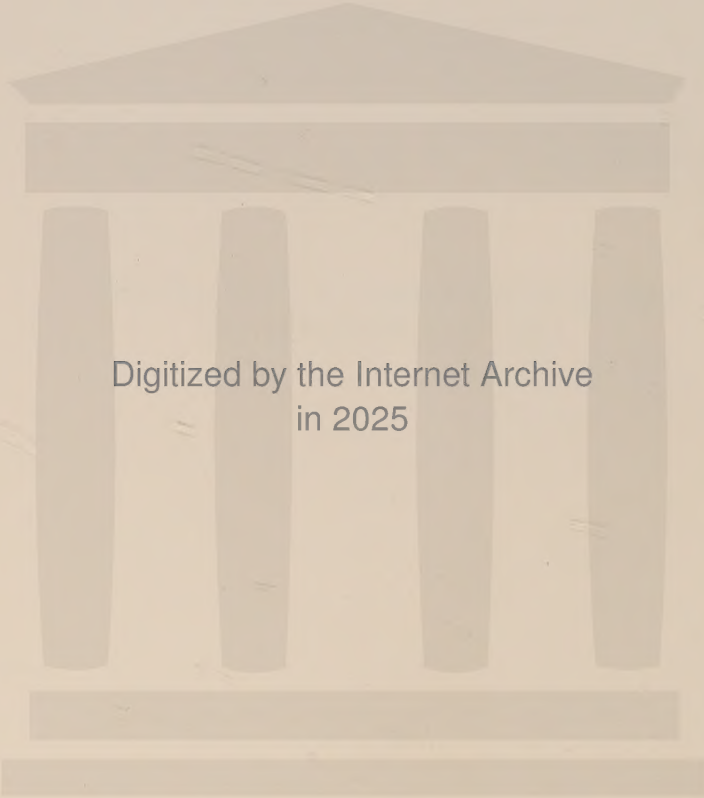


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THE MECHANISM OF THE HEART
AND ITS ANOMALIES



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THE MECHANISM OF THE HEART AND ITS ANOMALIES

Anatomical and
Electrocardiographic Studies

By
ÉMILE GÉRAUDEL

*Chief of the Laboratory of the Faculty of Medicine
of the University of Paris*

TRANSLATED, WITH AN INTRODUCTION

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KEY TO THE ELECTROCARDIOGRAM"*

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DEDICATION

To Professor Vaquez with the Homage of the Authors

To seek to determine the normal functioning of the heart by a study of the anomalies of the cardiac mechanism is the object of this work.

These anomalies are frequently encountered in practice and present themselves in many forms, but their analysis has become possible only since the discovery made by Einthoven. This savant has given us an instrument, the electrocardiograph, which enables us to determine, with a neatness and precision previously unknown, the action of the auricle as well as that of the ventricle.

In order to draw from the abnormal an idea of the normal, it is evident that we must employ hypotheses. The classical theory appears to us to be open to criticism, and we propose to substitute for it that of the cardioneccors with variable circulation, which has seemed to us to account more satisfactorily for the facts observed. And already certain of its deductions have been confirmed by the anatomical facts.

In this book the reader will find both the documents and the theories. The documents consist of numerous electrocardiographic tracings, some clinical observations, and microphotographs of lesions found at autopsy. All these documents have been gathered in the cardiological service of Professor Vaquez who has done me the honor, during the past four years, of entrusting to me the direction of his electrocardiographic and anatomopathological laboratories. Such is the activity of this service that I have been able in a relatively short time to make a large collection of significant facts. The theories may fall, but the facts will remain, and of these every new attempt at explanation must take account.

The following is the general division of the work: First I give a description, as complete as possible, of the electrocardiographic principle and technique. Next in order I note the anatomical data which are indispensable in any study of the cardiac mechanism, and following this is a description of the normal electrocardiogram and its modifications.

Then I come to a study of the anomalies of the cardiac mechanism, giving with each type first the classical explanation, and after that my own. The reader will choose between them.

Professor Vaquez, in placing at my disposal all the resources of his service has supplied the material for this work. If for no other reason,

his name should be inscribed on the first page; but it is my earnest wish that he may see more than that in this dedication—I would have him find in it the gratitude of a devoted collaborator.

I wish to thank my fellow workers in Professor Vaquez's service—Assistant Professor Donzelot and Drs. Theodoresco, Bordet, Mouquin, Giroux, and Parvu. They have shown their interest in my researches by bringing to my notice the various curious facts coming under their observation.

I must not forget to mention Élie Connault, my faithful laboratory assistant for twenty years, to whom belongs the credit of having so well made all the histological preparations herein illustrated.

Finally it is my agreeable duty to thank MM. Masson et Cie for consenting to undertake the publication of this work, to which they have given much care and the benefit of their great experience.

ÉMILE GÉRAUDEL.

*Hôpital de la Pitié,
October, 1927.*

FOREWORD

In 1749 when entering upon the study of the structure of the heart, its action, and its diseases, Sénac expressed some doubt regarding the utility of the undertaking. "So many skilful hands," he said, "have rummaged in this machine that one can hardly hope to find anything that has escaped them." However, what encourages one is that even the most careful investigators leave behind them unperceived riches which others may gather; and that, to quote an ancient writer, "Nature can never be exhausted."

Sénac was right, for a hundred years later new ideas which he could not have even imagined arose to transform cardiology. We had learned to recognize the play of the valvular apparatus, the respective rôles of the myocardium and of the nervous system in the functioning of the heart, the mechanism and the significance of the normal and pathological sounds. It seemed as though this time the cycle was closed and that nature, after all, was at the point of exhaustion.

Wrong again! Matters had reached that point when, at the end of the last century, the discovery of a conductor system of motor excitation through the heart disturbed the calm of the physiologists who had convinced themselves of the exclusiveness of nervous excitation. In face of the new facts they had to recognize that this dogma was not infallible, and to grant an important place to the conduction system of myogenic nature in the cardiac automatism.

This fact established, everything at last seemed to be satisfactorily arranged; there was no more obscurity, and everyone unanimously agreed that a specific tissue, largely supplied with nervous elements governed the automatism and therefore held control of the cardiac rhythm; to the myocardium belonged the task of receiving the stimulus and transforming it into contractile energy; to the extra- and intra-cardiac nervous system that of enabling the heart to adapt itself to the needs of the organism.

If that were the case (and nothing seemed to justify further doubt), there was no occasion to look elsewhere for the cause of the rhythmic action of the heart and of its physiological modifications. It followed equally that pathological states could be due only to a disharmony between the different factors of the cardiac contraction.

But pathology had not said its last word. The extreme variety of cardiac

syndromes which it recognizes eludes simple explanations and disarranges the cleverest experimental combinations. It was soon evident that, although in the great majority of cases the preceding data justified the formulation of a rational explanation of the anomalies of cardiac rhythm, yet occasionally they were found to be absolutely insufficient for this purpose. Rather than admit this, and in order to force these, so to speak, rebellious cases to submit to the general law, certain authors constructed various hypotheses, the very multiplicity of which bore witness to their weakness. Others asked if it were not wiser to modify somewhat the generally accepted beliefs. Among those who have held this idea are my fellow workers and myself, as witness the book which I published in 1926, in collaboration with Donzelot, upon the disturbances of cardiac rhythm. No one, however, has shown better than Géraudel, the evident necessity of a reconsideration of this whole question.

While forbearing to make *tabula rasa* of the ideas so laboriously acquired regarding the mechanism of the heart's action, M. Géraudel calls attention to a serious omission, namely, that no account has been taken of the incidental rôle of the circulatory system, although Haller long ago had glimpsed this influence. His previous studies on the distribution of the arteries and veins in the various parts of the myocardial tissue had shown him the indisputable relation of the circulation with both the normal and the pathological functioning of certain of these regions, in particular of those which seemed to play the principal rôle in the rhythmical action of the heart. The examination of pathological cases which were apparently unique and inexplicable by previously accepted theories, confirmed him in these ideas, and it is these which he upholds in the present work—based not upon theoretical conceptions but upon anatomical and clinical findings which establish their solidity. This has led him at the same time to revise the terminology at present in use in regard to the various modalities of rhythmic troubles. This revision is pardonable in view of the fact that the author has felt himself forced toward a clearer terminology which will make for a complete understanding of the new interpretation which he offers of the mechanism of the heart and of its anomalies.

It is possible that exception may be taken to certain of M. Géraudel's opinions. However, the author does not expect to force the reader's acquiescence at the very start. He only asks him to weigh his arguments without prejudice, or, as Bacon says, to examine the documents which he submits "with an eye not moistened by human passions." His only ambition is to give the reader occasion for reflection and to convince him that, whatever one may think, the problem of the mechanism of the heart's action is not yet completely solved.

Whether or not we accept the solution which he offers of this question we cannot fail to recognize the qualities of which he has given proof in this work—originality of viewpoint, good faith, and a tone of conviction. This book will surely mark an epoch in the history of cardiology, and I confess to a certain pride in having been able to procure for M. Géraudel the means of bringing it to a successful conclusion.

H. VAQUEZ.

PREFACE TO THE AMERICAN EDITION

It has been our custom from time to time to undertake some medical literary enterprise. When the work of Emile Géraudel came to our notice we sought the author's permission to translate it. We believe that in doing this we are rendering a real service to the English speaking fraternity of cardiologists.

Géraudel's work is an example of a painstaking and detailed study of cardiac function. We are not in a position to affirm or deny the truth of his fundamental conceptions, nor are we able to predict whether they will all be proved or disproved. He has, however, made out a strong case for his hypothesis. Whether further researches will sustain his theories or not, the author has at least rendered a service to cardiology in directing attention to the possibly supreme importance of its intrinsic blood-supply in the normal functioning of the heart; and also in his suggestion of a vicious circle established by circulatory disturbances in the sino-auricular node or the bundle of His—the cardionectors as he calls them.

Imagination is a faculty fundamental to progress. A man thoroughly familiar with the present state of the knowledge of his subject, who projects his imagination from the known to the unknown, forming the best hypothesis that he can, and who then throws all his energy into the affirmative side of the debate that is thus aroused, gives a real stimulus to progress. Research departments that are now maintained by all the great enterprises in the world have led to an astonishing progress of civilization in bringing material comfort to human beings throughout the world. Imagination is a foundation of research because research must be primarily a child of the brain, however much it may later be materialized in the test tube or under the microscope.

We are well aware of the difficulty of this translation, particularly as the author has given new meanings to words, even in French, and we would advise the reader, however slight may be his knowledge of French, to read the English translation with the French original at his elbow. A French word is not translated when its English analogue is used.

We owe a vast debt of gratitude to our lifelong friend, Dr. Thomas L. Stedman, who has been associated with us in this work; to Dr. Edward Preble, who did much of the labor of translating; Dr. Gertrude H. B. Nicolson, who has reviewed with us the section on electrocardiography; Mr.

Albert Gaudin of the Boulitte Company, long closely associated with us, who helped us with the translation of the description of the electrocardiograph; to our own office staff, and to the publishers for their valued co-operation.

The elder writer was guilty himself about twenty years ago of publishing a book on Arteriosclerosis in support of an hypothesis, which, strangely enough, even at this late day, is neither affirmed nor denied, so he is highly sympathetic with Dr. Géraudel in his pioneer undertaking.

LOUIS FAUGÈRES BISHOP.

LOUIS FAUGÈRES BISHOP, JR.

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PART I

ELECTROCARDIOGRAPHY AND ANATOMY
OF THE HEART

CHAPTER I

ELECTROCARDIOGRAPHY

Electrocardiography has as its aim the graphic interpretation of the variations of the electrical phenomena which accompany the functioning of the heart. The curve thus obtained is termed the electrocardiogram.

PRINCIPLE OF THE METHOD

If we introduce into a closed electric circuit a source of electromotive force, together with a current detector, every variation of electromotive force which is produced at the source will be shown by a variation of the current which the detector will reveal.

Now the heart is a source of electromotive force. A. D. Waller¹ in 1887 showed that one could introduce into the circuit, in place of the heart, the subject himself, the electrical variations of the heart being then transmitted to the periphery of the body. This method, at first employed only by physiologists, later became of service to clinicians. In the practice of *direct* electrocardiography, as employed by physiologists, the electrodes were brought into contact with the heart, but in *indirect* electrocardiography, as employed clinically, the electrodes are applied to the skin of the subject.

The intensity of the current is minimal, so that a sensitive detector is necessary. Waller used the capillary electrometer of Lippmann, but in 1903 Einthoven² devised the string galvanometer, and thanks to this apparatus electrocardiography became at once of practical service.

Figures 1 and 2 are taken from a brochure of Einthoven's.³ The former is a reproduction of an electrocardiogram of Lead I, taken June 15, 1894, by means of the capillary electrometer of Lippmann; while figure 2 is a reproduction of the electrocardiograms taken in the three Leads I, II, III, 31 years later (1925), from the same subject, with the string galvanometer.

The essential part of Einthoven's galvanometer is the string, that is to say a conduction wire of extreme tenuity, 0.002 to 0.003 mm. This string is stretched vertically between the poles of a powerful electromagnet, in such a manner as to be brought within a very intense magnetic field. If a current, be it ever so feeble, such as that of the heart, traverses the string, the latter is deflected from its position of equilibrium and by its greater or less displacement indicates the strength of the current which traverses it.

Since the weight of the string is very slight and in its movement it encounters no resistance save that of the air, it can be readily understood how rapid its response will be and with what fidelity it will follow the electric variations.

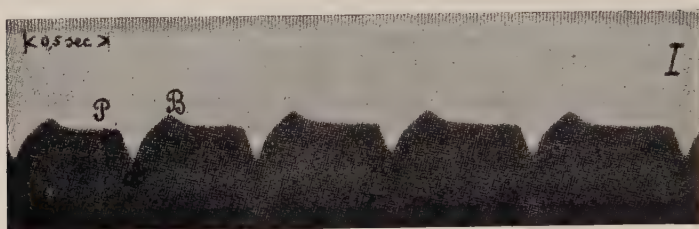


FIG. 1

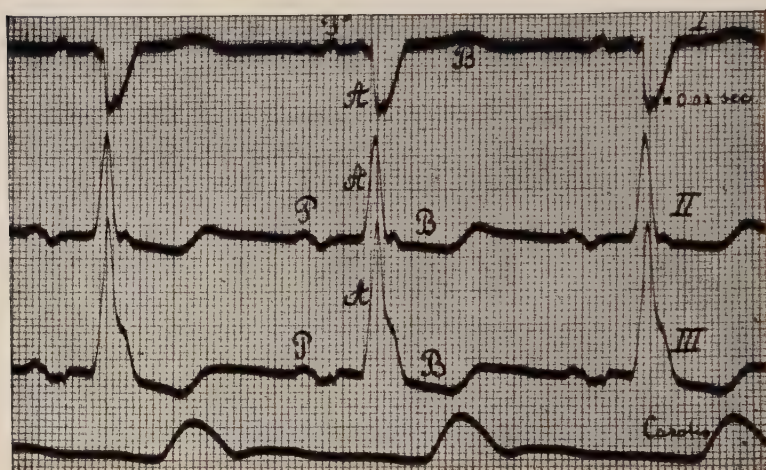


FIG. 2

FIGS. 1 AND 2. Two tracings taken by Einthoven from the same subject at an interval of forty-one years; the first was recorded by the capillary electrometer of Lippmann, the second by Einthoven's electrocardiograph. Compare the tracing in figure 1 and the upper one in figure 2, both taken with the same Lead I. The two tracings have the same physiognomy. (Einthoven.)

TECHNIQUE

Numerous types of apparatus have been devised on the principle of the string galvanometer of Einthoven, but we make use of that made by the firm of Boulitte (fig. 3) in the construction of which M. Bull, sub-director of the

Marey Institute has aided by his advice. Its clinical employment has been very extensive in the service of Professor Vaquez at the Pitié Hospital, Paris. We will not give a detailed description of the apparatus here for this will be found in the literature of the manufacturers. It seems better to us to explain merely the general scheme.

THE ELECTROCARDIOGRAPH

In every electrocardiograph there are two circuits, principal and secondary. The former is the galvanometer circuit which may be completed

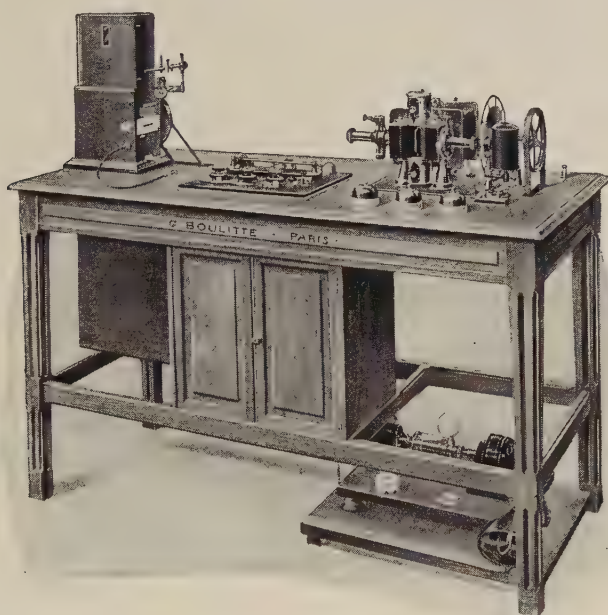


FIG. 3. The Boulitte electrocardiograph

either by the introduction of a fixed resistance of 700 ohms, or by that of the patient. If the latter is introduced into the circuit by the two upper extremities the lead is termed L. I; if by the right upper and the left lower extremity, L. II; and if by the upper and lower extremities on the left side, L. III (figs. 4, 5, 6). A special distributing commutator, D, permits the immediate substitution in the fixed resistance of any one of the leads, (L. I, L. II, L. III). These three were chosen originally as a result of Waller's researches. Under exceptional conditions and for special reasons we

may utilize other leads, such as different points in the precordial region, the anterior and posterior aspects of the thorax, the esophagus, the anus, etc.

This principal circuit may be made or broken by means of a contrivance called a shunt (figs. 4 and 7), which will be described later.

The secondary circuit contains a standard battery of which the constant



FIG. 4. The switchboard. Along the upper border of the figure are disposed from left to right of the reader, the two terminals of the standard cell; the three with which are connected the electrodes placed on the right arm (BD), the left arm (BG) and the left leg (JG) respectively, and the two terminals connected with the two ends of the string. Below these is the rheostat, R, with its slide pushed to the left. On the lower row, from left to right, are the potentiometer with its reversing switch, I; the shunt, S, affording protection to the string; a special form of commutator, D, which allows any one of the three leads to be switched on to the galvanometer; RF, the fixed resistance; I, II, III, the three leads; and the Morse key, C.

electromotive force is one volt, but it allows of a series of resistances, as follows:

1. A fixed resistance of 4745 ohms.
2. A resistance of 250 ohms which may be varied since it is the resistance produced by a sliding rheostat.
3. A resistance of 5 ohms which may be cut out or reintroduced by means of a short circuiting (Morse) key.

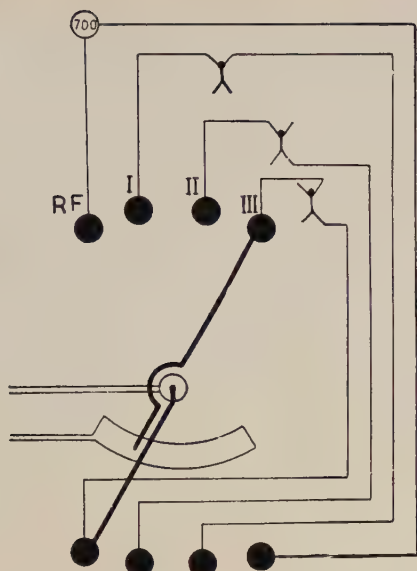


FIG. 5

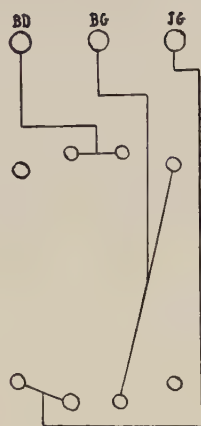


FIG. 6

FIG. 5. Detail of the commutator for the leads, showing how, in moving the button D on the switchboard one closes the principal circuit of the galvanometer by the introduction of either the fixed resistance of 700 ohms or of the patient in Lead I, II, or III.

FIG. 6. Detail of the connections. This plan shows how the contacts of the commutator of the leads are connected with the terminals BD, BG, and JG, in relation with the electrodes placed on the limbs of the patient.

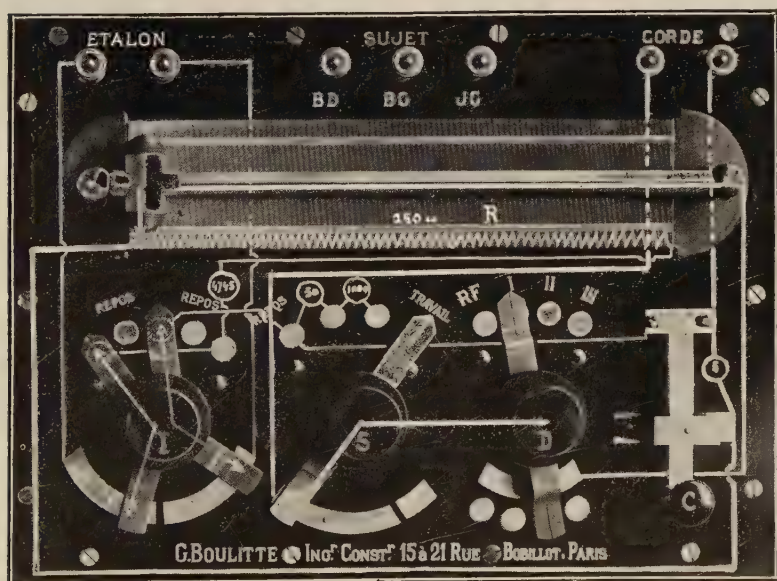


FIG. 7. Detail of the electrical circuits of the switchboard. The principal circuit is represented by a thick line, the secondary circuit by a thin line. The circles represent the resistance coils with their value in ohms.

The principal circuit and the secondary circuit have a common switch which may close one or the other, or the current may pass through both circuits together.

Rôle of the Two Circuits

The rôle of the principal circuit is simple. It contains the galvanometer and the patient; the result of this is that every electric variation of the patient is manifested by a deflection of the galvanometer string.

The rôle of the secondary circuit is double: 1. That of neutralizing the current of the skin; 2. That of adjusting the string.

1. By the skin current we understand all currents not originating in the heart which are produced with the patient at rest. It is conceded that these currents are almost entirely produced by the functioning of the glands of the skin. The skin current varies with each individual and it is compensated for by a current of equal intensity and of opposite direction which is thrown into the circuit. The proper intensity of current is obtained by varying the resistance of the rheostat by moving the slide. The proper direction is obtained by means of an apparatus called the current reverser.

2. We have seen that the battery has an electromotive force of one volt. We have also seen that the resistances have a total value of 5000 ohms (4745 plus 250 plus 5). From these data we learn that the battery current has an intensity of 1/5000 ampere, as shown by the well known formula, I

$$(\text{ampere}) = \frac{E (\text{volt})}{R (\text{ohm})}.$$

On the other hand from the upper to the lower limit of the resistance of 5 ohms, which the short-circuiting or Morse key (C) controls, there is a drop in the electromotive force called the ohmic drop, the value of which is one millivolt, according to the formula, $E = RI$, that is to say $E = 5 \times \frac{1}{5000} = \frac{5}{5000}$ volt. From this it follows that when we cease to short circuit the resistance by pressing the key we introduce this resistance of 5 ohms into the circuit, and the electromotive force varies by one millivolt. The string then, through its deviation registers this variation of the electromotive force. From now on it is easy to regulate mechanically the tension of the string in such a manner that the deflection of its image will be one centimeter for each variation of a millivolt. Thus the string will be standardized in conformity with the international agreement, permitting the comparison of tracings from different sources.

The Shunt

This contrivance enables us, when the lever is in what is called the rest position, to break the principal circuit. The string is then shut out from

the current proceeding from either the patient or the battery. A shunt is a diversion established in a circuit whereby a given fraction of the main current is allowed to pass through a parallel or derived circuit. We know that when two circuits of different resistance are offered to the passage of an electric current the latter divides into two parts, each of which has a value inversely proportional to the resistance.

When a patient is brought into the principal circuit we are unaware of the strength of the electromotive force which is about to act on the fragile string. It is therefore necessary to pass gradually from the rest position to that of activity obtained by contact of the lever with the fourth and last stud, while increasing progressively the intensity of the current. We "shunt the string."

When the lever passes to the second stud the main circuit is closed so that the current of the patient may pass through the string. But at the same time we establish a diversion of this current into a circuit which does not pass through the string. Now in this circuit there is a resistance calculated at 50 ohms, and the resistance of the string being about 1500 ohms, we see that the current from the patient is divided between the two circuits, 29/30 of the current being diverted and 1/30 passes through the string. The string undergoes a slight deflection under the action of the skin current, a deflection which is corrected by compensating the skin current. On the other hand it also presents fine oscillations under the action of the heart which indicates that the connections are good and that the electrocardiograph is functioning. When the lever is passed to the third stud we introduce into the diverted current a supplementary resistance of 1000 ohms which makes a total of 1050 ohms. The current of the patient is then divided in a different fashion, two-thirds being diverted while one-third passes through the string. The deflection of the skin current due to the string is increased, but is easily corrected. The displacements of the string of cardiac origin are wider.

In placing the lever on the fourth stud in the working position, we suppress the shunt. All the current which comes from the patient passes through the principal circuit and the skin current is perfectly compensated. The cardiac current acts alone and in its totality upon the string of the electrocardiograph.

The scheme of the two circuits is shown on the switchboard of the Boulitte apparatus (fig. 7). We see here the anatomy of the apparatus and readily comprehend what takes place when we manipulate the rheostat, the inverter, the interrupter, the shunt, the distributing commutator and the Morse key.

The Optical Apparatus

The string (fig. 8) is of such minimal calibre and its deviations are so slight that they are not appreciable to the naked eye. An optical apparatus, actually a horizontal microscope, projects the enlarged image of the string



FIG. 8. The galvanometer string. The string is carried in a holder which is inserted between the poles of the galvanometer. The tension of the string may be modified by means of the milled head, B, which turns the micrometer screw, V, and the lever, L.

on the screen (fig. 9). The image is enlarged 500 times, the screen being at a distance of 75 cm.

The Photographic Chamber

Behind a slot in the screen, there is unwound at a constant speed a roll of sensitive paper upon which is projected the image of the string. At the

speed habitually employed in the tracings which we publish, 5 cm. of film are unrolled in one second (fig. 10). For research work, four different speeds can be obtained. The paper can be adjusted to unroll at 12.5 mm., 25 mm., 50 mm., or 100 mm. per second.

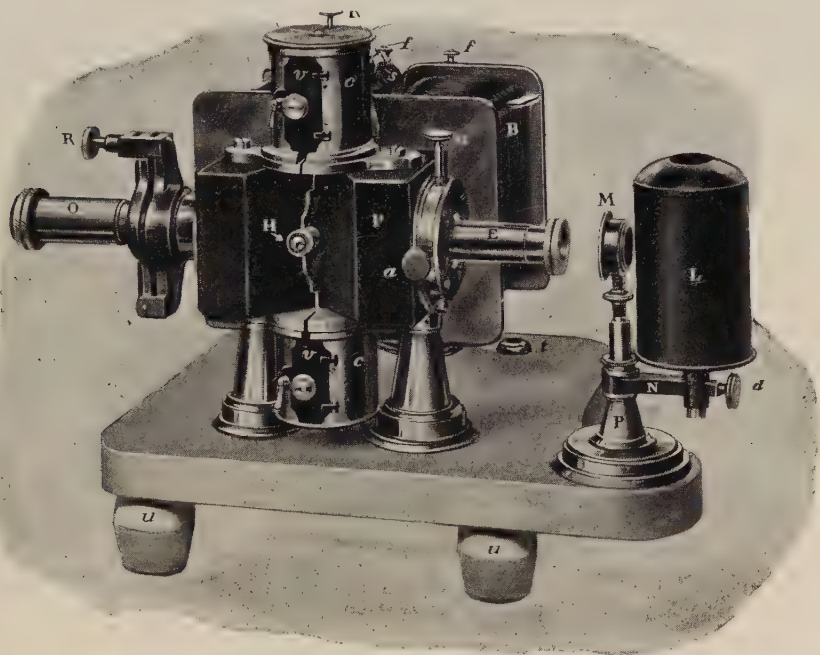


FIG. 9. The galvanometer and the optical apparatus. The string in its holder is in place between the two poles, P, of the electromagnet. These pole pieces are bored to receive the tubes of the horizontal microscope; one tube, E, contains the condenser, the other tube, O, carries the objective and eye-piece. The focussing of the image is done by the micrometer screw, R. The incandescent lamp is enclosed in the lantern, L. M, a condensing, or rather a field-lens.

The Chronograph

This consists of a rotating disk with 10 projecting spokes which shut off the light every fiftieth of a second. The time is thus registered upon the curves by fine lines which traverse the entire width of the sensitive ribbon. Two of the spokes, diametrically opposite each other, are wider than the others. They cause on the tracings a slight increase in thickness amounting to 1–10 second. One millimeter corresponds to $\frac{1}{50}$ or $\frac{2}{100}$ of a second, if the film travels at a speed of 5 cm. a second, (or $\frac{1}{25}$ second when the speed is 2.5 cm. a second).

To determine rapidly the rhythm of an electric impulse we take the sum

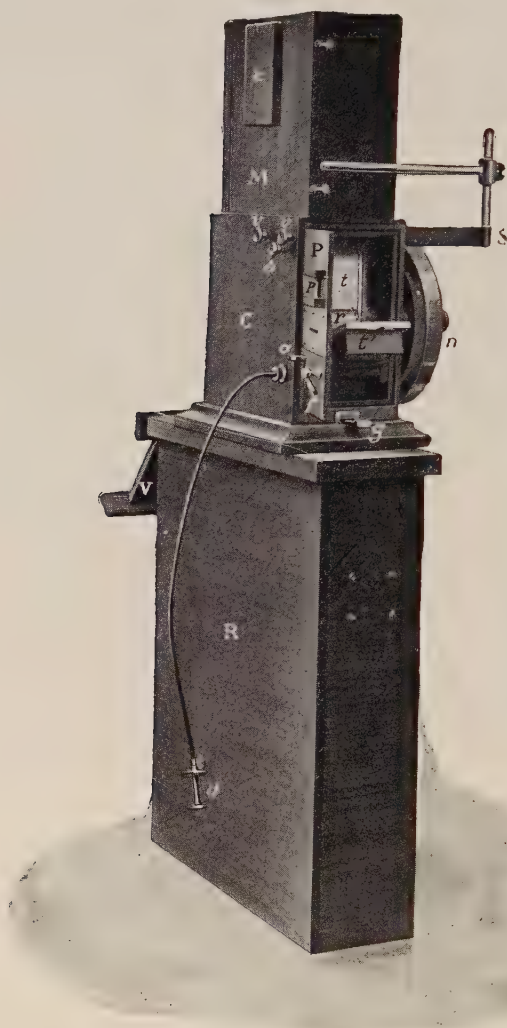


FIG. 10. The photographic recorder, M, a box containing the reserve of sensitized paper or film. C, the camera, properly speaking, containing the mechanism for moving the film which is drawn from the upper compartment, through the passage, t, by the sprocket-wheel, r. On the right, between o and p, is a narrow horizontal slit through which the image of the string is thrown and photographed. Pressure on the button, g, pushes a knife which divides the film on a level with the lower part of the camera; the film falls into the lower compartment, R, which is closed by the shutter, V, and the film is carried to the dark room for development. On the glass plate, P, details of name, date, etc. are written and photographed on the beginning of the film.

of the intervals between the fine lines of the chronograph which separate two of the consecutive accidents. We then divide 3000 by the total number of these intervals, the quotient being the rate of the rhythm. Thus between two peaks on the tracing there are 30 intervals; the quotient of 3000 divided by 30 gives us the rate which is 100.

A more convenient procedure consists in making use of a sort of rule on which are inscribed rhythms corresponding to fixed lengths. I have had the Boulitte firm make a model of this "rhythmometer." One may see upon the tracings which we publish that the longitudinal lines on the film form squares by intersection with the lines of the chronograph. The finer lines are 1 mm. apart, the thick ones occur at every half-centimeter. These lines are marked on the film as it unrolls before a cylindrical lens on which corresponding lines are engraved. They facilitate the measurement of the heights of the peaks and the corresponding value in millivolts.

TAKING AN ELECTROCARDIOGRAM

Adjusting the Electrodes

We have abandoned the use of receptacles into which the patient introduces the two hands and the left foot and employ in place of them electrodes made of thin plates of tin enclosed in a flannel cover which is rolled around the limb. This band is wet with a saline solution, 20 per cent, which should be hot. It is a good practice to wet it frequently. Almost the entire difficulty in the use of the electrocardiograph has to do with the electrodes. Keeping them in good order and applying them correctly are therefore two points of great practical importance.

Inscription of the Name

First we must be sure that the box from which the film is unwound is in position, with its shutter open. It is convenient to photograph on the beginning of the film the name of the patient, his number, the date, etc. These are written on a glass slip by means of India ink slightly moistened with water, and a simple rinsing with water serves for their subsequent erasure. This slip is placed in front of the shutter. The inscription is made in five stages:

1. Light the lamp.
2. Let the string drop from in front of the shutter mechanically, by loosening one of the tension screws. In this way we prevent the image of the string from obscuring the inscription.
3. Make the photograph on the film by opening the shutter.

OPERATION OF THE ELECTROCARDIOGRAPH

Film Receptacle Opened

Preliminary Operations

Open the Circuit:—of the Lamp, of the Galvanometer, of the Battery

First Stage	Second Stage	Third Stage
Turn the lead switch (D) to RF (fixed resistance) Turn the shunt switch (S) to <i>travail</i> (activating the string) Adjust tension of the string (to about 1 mv. = 1 cm.)	Turn lead switch (D) to I (II, III) (bringing the subject into the leads) Turn shunt switch (S) gradually to <i>travail</i> (Compensating the skin current at each contact) Adjust tension of the string (to exactly 1 mv. = 1 cm.) Taking the Tracing Focus the string Start the film unwinding Juggle away the exposed portion Cut the film	Turn lead switch back to RF (fixed resistance) Turn shunt switch rapidly to <i>travail</i> (activating the string) Adjust tension of the string (to about 1 mv. = 0.5 cm.)
Turn shunt switch to <i>repos</i> (deactivating the string)	Turn shunt switch to <i>repos</i> (deactivating the string)	Turn shunt switch to <i>repos</i> (deactivating the string)

Final Operations

Close the Circuit:—of the Lamp, of the Galvanometer, of the Battery

Film Receptacle Closed

4. Restore the string to its place before the center of the shutter.
5. Extinguish the lamp.

Moving the Used Portion of Film away from the Shutter. The shutter is situated about 5 cm. above the slot through which the photographic record of the deflections of the string is taken. To make sure that this portion of the film is not impressed anew in passing the slot, it is moved along for a distance of 5 cm. with the lamp extinguished; this is equivalent to allowing it to unwind for one second.

Manipulation of the Electrocardiograph

The current having been established in the respective circuits of the lamp, the galvanometer, and the battery, this manoeuvre is accomplished in three phases.

In the first the principal circuit is completed by the fixed resistance—position of departure.

In the second the principal circuit is completed by the patient, substituted for the fixed resistance, in Leads I, II, and III—position of activity.

In the third the principal circuit is completed by the fixed resistance, substituted again for the patient—position of return.

One must never replace the fixed resistance by the patient or the reverse, nor should one pass from one lead to the other, without breaking the principal circuit. It is indeed necessary, whenever we would modify the intensity of the current passing through the string, to shield the latter from too sudden a change.

Each of the three stages then is always terminated by the same tempo, namely breaking the circuit of the string.

In each of the three there are three successive operations, always the same and always in the same order. These are: (a) Putting the string into action by closing the circuit; (b) Regulating its tension; (c) Putting it at rest by breaking the circuit.

Three stages then and in each stage three operations. The accompanying table summarises the stages and operations. We will explain briefly each of these phases and operations.

First Stage. The three operations of this phase take place upon fixed resistance, the electromotive force of the patient does not interfere and the only force in play is that of the battery. The current generated is fixed and its intensity is known; one may therefore pass it in full strength through the string without special precaution.

(a) The string is put in action and from the position marked “repos” on the control board, we pass directly into the position of activity (“travail” on the control board) without any previous shunting.

(b) The string is put in tension and this should be such that its displacements under the action of current variations will have a readily appreciable amplitude. A good tension is obtained when the displacement of the string is from one centimeter to one and a half centimeters for the variation of a millivolt, a variation obtained by depressing the Morse key.

(c) The string is brought again to rest by breaking the principal circuit.

Second Stage. We substitute for the fixed resistance the patient introduced into the principal circuit in Leads I, II, III. When the circuit is closed the string is submitted to an unknown electromotive force, combined with an unknown resistance. We must not then throw the whole of the unknown current abruptly through the string.

(a) We then pass progressively, contact by contact, from the position of rest (*repos*), when no current passes through the string, to the position of activity (*travail*) when the entire current passes through it, deriving the greater part of the secondary current at the second contact and a smaller part at the third contact. Moreover, as the quantity of the skin current of the patient, which passes through the skin, increases, when we pass from one contact to the next, the deflection of the string should be compensated at each contact by means of the rheostat and inverter. At the "travail" stud we make the final compensation.

(b) Putting the string in tension is necessarily a very delicate operation. A variation of one millivolt should cause the image of the string to deviate exactly 1 centimeter.

(c) Putting the string at rest is effected exactly as in the first phase.

Taking the Tracing

It is between the two operations (b) and (c) that we take the tracing. Occasionally we may omit the tracing, merely following the displacements of the string with the eye. We may, in this way, diagnose numerous anomalies of rhythm, and it is also good practice for the eye for it enables one to take account of the type of electrocardiogram with which one is dealing and also to decide upon the moment at which to begin the tracing, and upon the length of tracing required.

Two points should be borne in mind, namely (1) to have the image of the string exactly in focus so as to obtain a tracing with distinct outline, and (2) to unwind the film behind the slot.

Moving the Exposed Portion of the Film From in Front of the Slot

The slot is about 12 cm. above the guillotine which cuts the film, so that there are 12 cm. of film already used between the slot and the guillotine.

We should therefore roll off 12 cm. of ribbon beyond the guillotine; this takes about three seconds when the film travels at the usual speed; the knife will divide the film at a point where it has not been exposed.

Third Stage. We again substitute the fixed resistance for the patient.

(a) The string is first activated, as in the first phase.

(b) The string is then stretched, the object of this being to ensure a proper tension in order to avoid rupture. The string must not be too taut for the least increase caused by a change of temperature or moisture will add to the risk of breaking it; nor should it be too slack, for then there will be the risk of its adherence to one of the poles of the electro-magnet, with a new possibility of rupture when it is brought into action. A good resting tension is attained when a deflection of the image of half a centimeter corresponds to a variation of one millivolt.

The three stages being finished we proceed to the final manipulations, which correspond to the preliminary ones and consist in breaking the respective circuits of the lamp, the galvanometer, and the battery. The case containing the used length of film is closed and taken to the dark room where the film is developed.

Thus regulated, stage by stage, the manipulation of the electrocardiograph is accomplished with the greatest ease.

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CHAPTER II

ANATOMY OF THE SPECIFIC MYOCARDIUM

THE CARDIONECTORS

The mechanism of the heart presents one of the most difficult problems in physiology, and it is far from being solved. Every attempted solution of it should take into account a certain number of anatomical data which are of relatively recent discovery. The heart comprises, aside from the contractile myocardium properly speaking, certain well localized areas of myocardial tissue, which in all probability exert a marked influence on the functioning of the contractile myocardium. This special tissue is usually called the "specific myocardium." There is a specific myocardium in relation to the contractile myocardium of the auricle, and another in relation to the contractile myocardium of the ventricle. I propose to give to the first name of atrionector and to the second the term ventriculonector. The two comprise the cardionectors.¹

The cardionectors are situated in the right auricle and it is solely in this cavity that one need search for the specific formations which control the movements of the heart. This fact will cease to cause us any astonishment as soon as we arrive at an exact conception of the right auricle. From the point of view of macroscopical anatomy the right auricle is one of four cavities of the heart, and its preeminence is not explained. But taking a true anatomical point of view we see that under the term right auricle we group structures of very different significance. The right auricle in fact consists of a median part toward the interauricular septum, and a right lateral part. These two portions communicate freely through a large orifice limited above and behind by the crista terminalis, a well-developed muscular arch, and below and in front by the valve of Eustachius and the valve of Thebesius. These three structures represent the different portions of the right primitive valve of the venous sinus.

The median portion corresponds to the sinus reuniens and the atrial canal; the first named is formed by the confluence of the two canals of Cuvier, the right and the left. The right canal of Cuvier becomes the superior vena cava and the left one becomes the coronary sinus. The atrial canal or primitive auricular canal follows the sinus reuniens and continues into the ventricle, into which it is slightly invaginated. The lateral portion

is an out-pouching of the atrial canal, in which are developed the pectinate muscles which are inserted on the crista terminalis. The left auricle is an homologous out-pouching from this same atrial canal, communication with which is usually closed at birth although it is free during intrauterine life and persists at times into extrauterine life as the foramen of Botall.²

The right auricle then is not the counterpart of the left; it comprises primarily a part which corresponds to the primitive vascular tube between the afferent veins and the chamber of ventricular expulsion, and forms secondarily an annex (the right auricle properly speaking). The other annex, alone, forms the left auricle.

The cardioneurotomes are found at the level of the parts which correspond to the primitive cardiac tube. To homologize exactly the portions of the primitive cardiac tube with the fully developed heart remains difficult, despite the labors of embryologists. Moreover, a certain incertitude exists when we come to name the cardioneurotomes. Keith and Flack³ have applied the term sino-auricular node to the structure they discovered and which we have designated under the name atrioneurotome. Léon Stiénon⁴ has rightly said that this node does not correspond at all to the sino-auricular canal of the lower vertebrates, or to the zone of junction between the sinus reuniens and the auricular canal. It is actually situated at the opening of the right canal of Cuvier (superior vena cava) into the sinus reuniens.

The other specific structure, the ventriculoneurotome, is situated, as we shall see later, in front of the opening of the left canal of Cuvier, the coronary sinus. There is thus a remarkable symmetry between the two. This also enables us to understand better how the cardiac nerves, pneumogastric and sympathetic, on the right side more readily influence the atrioneurotome, a structure near the right canal of Cuvier, while those on the left side act preferably on the ventriculoneurotome, a structure situated near the left canal of Cuvier.

The invagination of the sinus reuniens and the auricular canal into the auricles is much more marked on the left side than on the right, to such a degree that on the right side the wall of the sinus and its right valve are well developed in the completely formed heart. On the left side, on the contrary, the homologous portions are reduced to a minimum. Nevertheless we find that the atrioneurotome is situated between the right canal of Cuvier and the sinus reuniens and the ventriculoneurotome between the left canal of Cuvier and this same sinus reuniens.

The separation of the left auricle on the one hand, and the conjoined right auricle and atrial canal on the other hand, is a secondary matter. All of

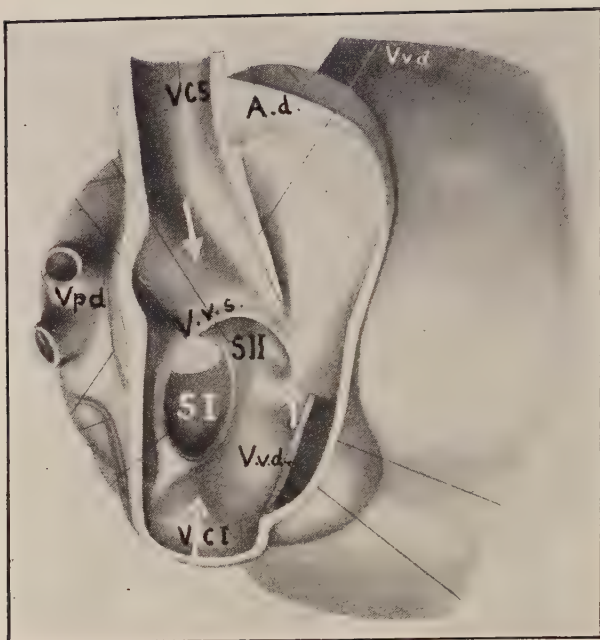


FIG. 11 . Interior of the right auricle of a human embryo, 36 mm. in length

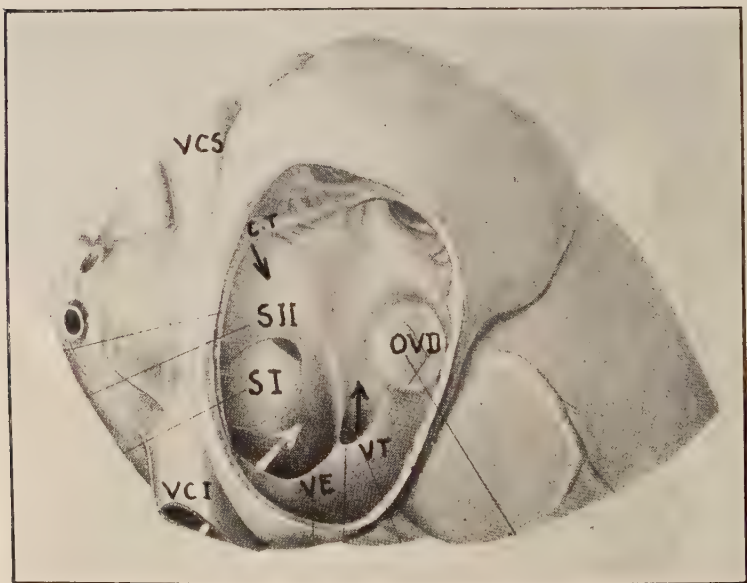


FIG. 12. Interior of the right auricle of a human embryo 310 mm. in length

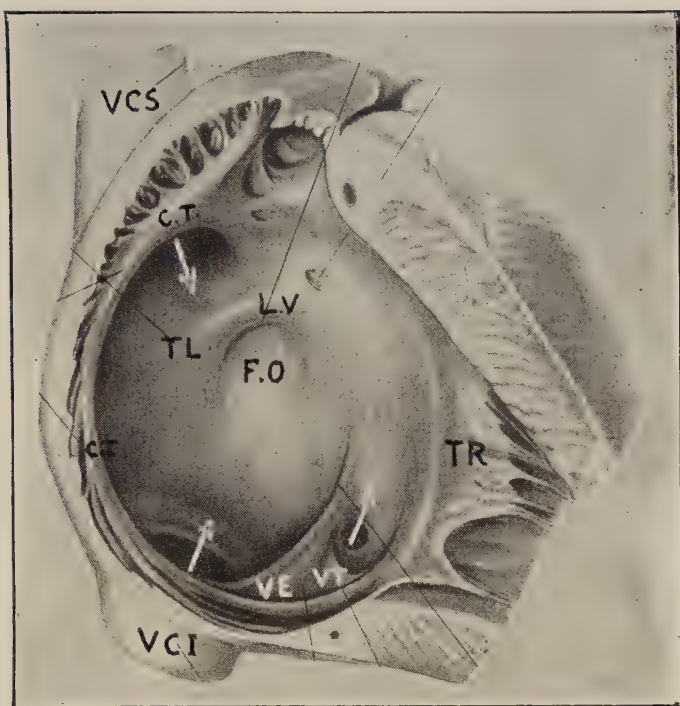


FIG. 13. Interior of the right auricle of an adult

FIGS. 11, 12, AND 13. The fully developed right auricle is formed by the union of the primitive right auricle, Ad, and the venous sinus, the latter being bounded by two valves—left, Vvs, and right, Vvd.

The figures, from Tandler, show different stages of the development of these parts. The figures are, however, not entirely comparable, for the models of Born, after which they are drawn, were cut in such a way that the excised part in figure 11 was much greater than that in figure 12. But on the other hand figure 12 corresponds exactly to figure 13. Therefore one must note, especially in following the evolution of the valves of the venous sinus, that the right valve is shown in its entirety in figures 12 and 13, while in figure 11 the greater part of the valve has been cut away, its upper and lower ends alone being shown.

With this explanation in mind: (1) We see how the separation between the two auricles is effected. It results from the development of the two septa, S I and S II, which close like the shutter of a photographer's camera, forming the fossa ovalis, F. O., and the limbus of Vieussens, LV. (2) We see what transformation the two valves of the sinus undergo: the left disappears; the right gives origin above to the crista terminalis, CT, and below to the valves of Eustachius, VE, and of Thebesius, VT. The first bounds the opening of the vena cava inferior, VC I, the second that of the coronary sinus. Vpd, right pulmonary veins; OVD, ostium venosum dextrum or tricuspid orifice; TL, torus or tubercle of Lower.

these structures constitute one whole—the venous chamber. Likewise the two ventricles are the result of the secondary division of a cavity primarily single—the arterial chamber. From the general viewpoint we have to consider not four cardiac cavities, but two—one auricular or atrial, the other ventricular. Electrocardiography has confirmed this view. Exact researches, in particular those of Athanasiu,⁵ have shown that there is a perfect synchronism in the contractions of the two auricles and in those of the two ventricles.

A description of the two cardionectors is now in order. I will begin by describing the ventriculonector, because all of its constituent parts have been determined, which does not seem to me to be the case yet with the atrionector.

THE VENTRICULONECTOR

Under this heading I propose to designate the compound structure formed by the node of Tawara, the bundle of His, and the network of Purkinje.⁶ These three formations were discovered separately, Purkinje discovered his fibrillary network in 1845; in 1893 the younger His⁷ described the muscular bundle between the auricle and ventricle which goes by his name; and finally in 1906 Tawara⁸ showed that the bundle of His bulges out above, at least in the dog, sheep, and cat, forming an oval nodule (now called, after the discoverer, the node of Tawara), and is continuous below with the fibrils of Purkinje. The entire structure—node, bundle, and network—has since that time been considered as a whole, and to it has been attributed the rôle of a conductor system of motor excitation from auricle to ventricle.

To clarify the description, it will be convenient to divide the ventriculonector into two parts—one condensed into the node of Tawara and the bundle of His with its two principal branches, and the other spread out into the network of Purkinje.

The condensed part of the ventriculonector lies in the septum which separates the two hearts, which septum has both an auricular and a ventricular portion. The interauricular septum is composed of two layers of muscular tissue, right and left, between which is interposed another layer of connective tissue, rich in fat. The interventricular septum is the result of the intimate contact of the two ventricular cones and is essentially muscular. But toward the base of the ventricles the septum becomes fibrous. In fact, on the side of the left ventricle, the muscular expulsive chamber becomes a subsigmoidal fibrous funnel, the zone of transition into the aorta.⁹ In the right ventricle also the wall ceases to be muscular. The septum in its fibrous portion thus constituted is called septum fibrosum or pars mem-

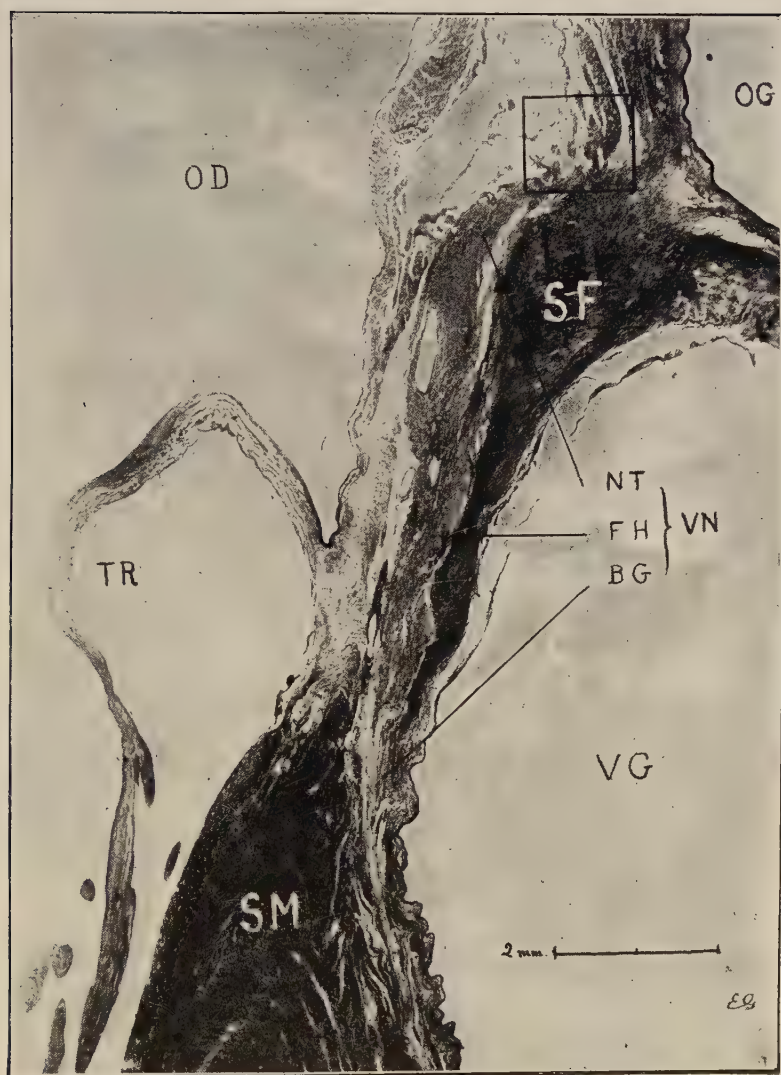


FIG. 14. The ventriculonector in a horizontal section of the heart, the apex of the organ pointing downward. VN, the ventriculonector formed of the node of Tawara, NT, of the bundle of His, FH, and of its branches, the left, BG, only appearing in the section. This formation is situated in the septum between the left ventricle, VG, and the right cavities separated by the tricuspid, TR. OD, right auricle, OG, left auricle, SF, the fibrous septum, SM, the interventricular muscular septum.



FIG. 15

FIG. 16

FIG. 17

branacea septi. This is the "undefended space" of the English authors and is very thin and translucent. The septal flap of the tricuspid valve is inserted in it. Above the insertion the septum separates the right auricle from the left ventricle. It is then, strictly speaking, interauriculo-ventricular. Below it separates the two ventricles and is interventricular (fig. 29).

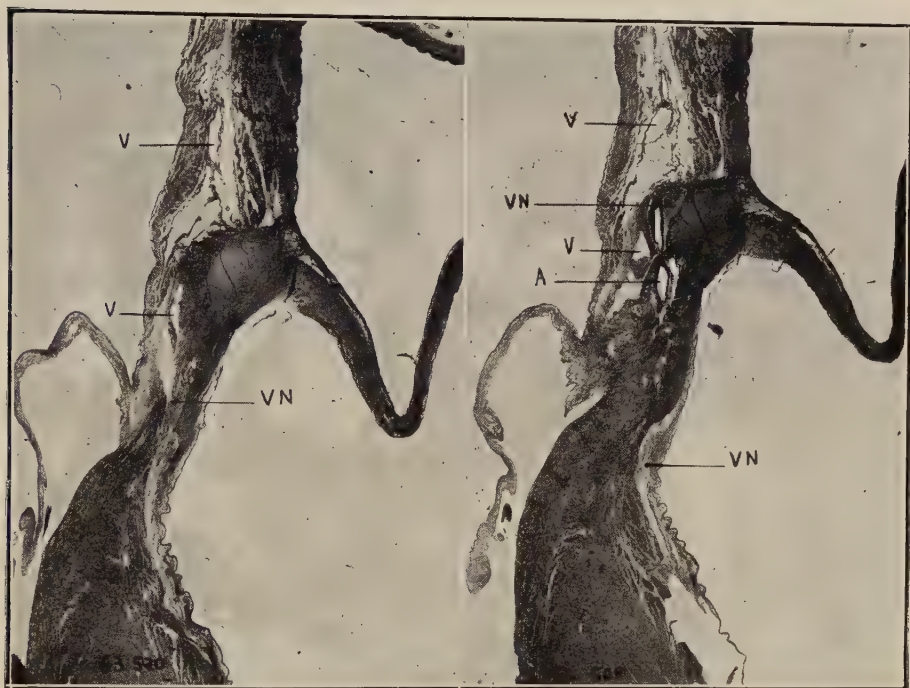


FIG. 18

FIG. 19

FIGS. 15 TO 19. Five horizontal sections of the ventriculonector. These five sections belong to the same horizontal series; figure 15 (section 160) is the highest of the series, the heart being with apex downward.

This borne in mind, the condensed part of the ventriculonector may be compared to a man stretched on his back. His head, the node of Tawara, is lodged in the adipose connective tissue layer of the interauricular septum; his torso, the bundle of His, lies in the interventricular septum at the junction of its muscular and fibrous parts; the back rests on the crest of the muscular wall, while the body is engaged in the fibrous wall. This imaginary man has no arms, but with his legs he bestrides the ventricular

wall, the right leg (right branch of the bundle) in the right, and the left in the left ventricle.

We will now discuss in detail the relations of these different portions.

The Node of Tawara

This is the auricular portion of the ventriculonector. It corresponds on the internal surface of the right auricle to the anterior angle of a triangle formed by the free border of the valve of Thebesius, the tendon of Todaro, and the adherent margin of the septal flap of the tricuspid valve, close to its union with the anterior flap. This is known as the triangle of Koch. The auricular face of Tawara's node is sheathed by myocardial fibers belonging to the right auricle, and toward its upper extremity is in contact with the fatty tissue in which is contained the vein of the ventriculonector. On the side of the left ventricle, Tawara's node corresponds to the most sloping portion of the insertion of the posterior semilunar valve—not the coronary—at one and a half millimeters below it. The ventricular face of Tawara's node is closely united to the fibrous septum, upon which it is moulded.

The superior and inferior faces of the node are in relation with the fatty tissue which separates the two myocardial sacs, in which run certain muscular fibers.

The Bundle of His

This prolongs without marked demarcation the node of Tawara. It is lodged in the interauriculoventricular fibrous septum. Its inferior aspect rests on the muscular septum, either on the crest itself, or on one of the flanks, right or left. After a short distance it divides into two branches. The right one is directed to the right, beside the right ventricle. There, for the space of a few millimeters it remains beneath the endocardium, and then sinks into the myocardium. It is lodged in the trabecula septomarginalis (supraventricular crest), a tract, or at times a muscular fasciculus which separates the ventricle, properly speaking, from the pulmonary infundibulum. The band in which it is enclosed forms a convex layer anteriorly and terminates at the anterior papillary muscles of the tricuspid. The right branch of the bundle of His continues beyond these under the form of a network of scattered fibers which will be studied later. In its end portion, before reaching the papillary muscles the right branch is again subendocardial. The left branch seems to prolong the bundle of His and is directed toward the left ventricle. It does not long preserve the cord-like aspect of the right branch, but soon spreads out into a fibrous sheet. This, however, is not continuous for soon after it arrives beneath the endo-

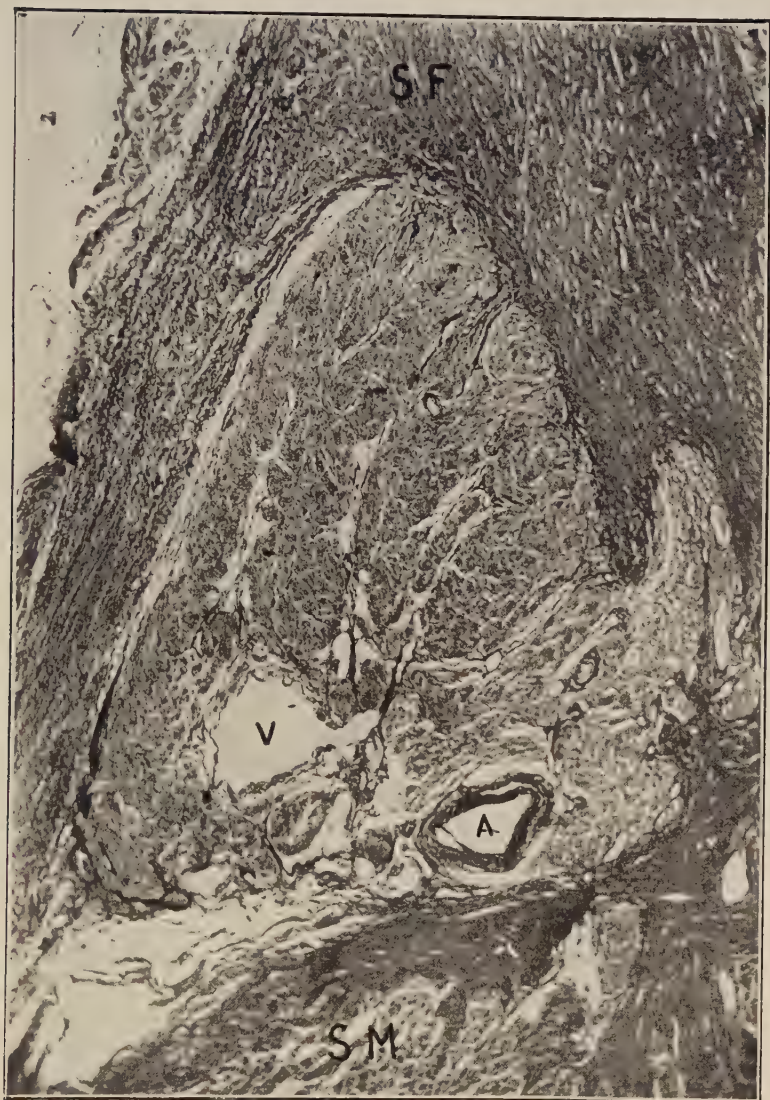


FIG. 20. The ventriculonector in frontal section. SF, fibrous septum. A and V, artery and vein of the ventriculonector; SM, interventricular muscular septum. (Magnified portion of a section very close to one seen under a low power in figure 22.)



FIG. 21

FIG. 22

FIG. 23

cardium of the aortic infundibulum, or expulsive chamber of the left ventricle, it subdivides into two parts, the anterior terminating at the anterior, the posterior part at the posterior papillary muscle, of the mitral valve. On leaving these it is continued by a network of scattered fibers in the same manner as the right branch.

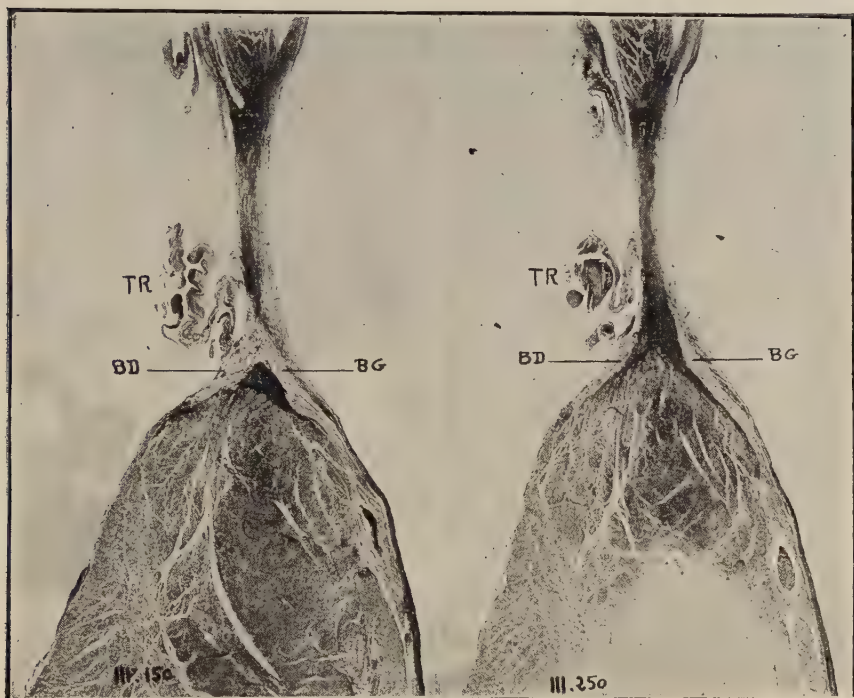


FIG. 24

FIG. 25

FIGS. 21 TO 25. Five frontal sections of the ventriculonector. These five sections belong to the same series passing from behind forward, the heart being supposed to rest with apex downward and facing the reader. VN, ventriculonector; BD, BG, right and left branches of the bundle of His; SF, fibrous septum; SIV, interventricular septum; MAD, myocardium of the right auricle; PA, aortic wall; TR, tricuspid valve.

The division of the bundle of His into two branches is not always accomplished in the manner just described. Fibers may be detached from the bundle to the right, then some to the left, and again others to the right, so that a frontal section will present the appearance seen in figures 21 to 25 inclusive. Since Tawara's discovery, further developed and confirmed by Mönckeberg¹⁰ who more particularly studied the human heart, to which we

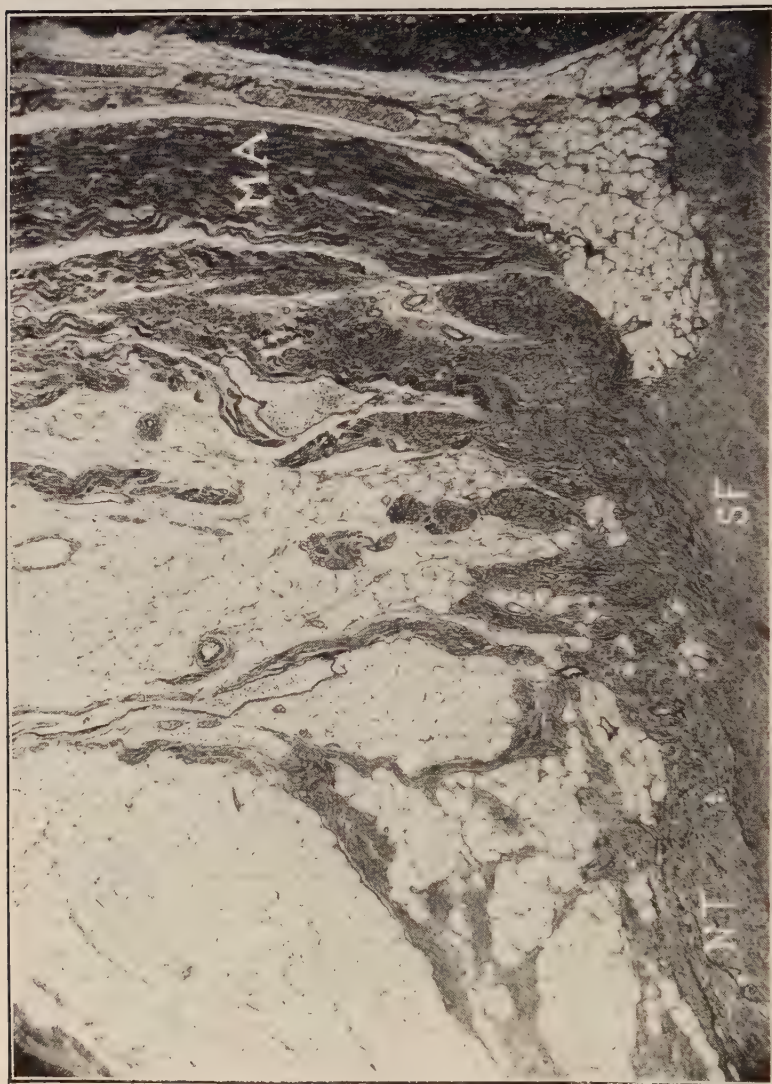


FIG. 26. Transition zone between the node of Tawara and the myocardium of the auricle. This figure is a magnification of the region enclosed in the square in figure 14. NT, upper extremity of the node of Tawara; MA, myocardium of the auricle; SF, fibrous septum.

also are limiting our researches, we know that the two branches of the bundle of His, after leaving the anterior tricuspid papillary muscles and the anterior and posterior ones of the mitral valve, are continued by a network of myocardial fibers, somewhat specialized, and long known as Purkinje's fibers. It is the network in its entirety that we have called the "dispersed portion of the ventriculonector."

Fibers of Purkinje

In the right ventricle the fibers of Purkinje diverge on leaving the anterior semicircumference of the anterior papillary muscle (pillar) of the tricuspid, following three directions which were well defined by Mönckeberg, whose description is as follows:

"A branch curves forward to gain the anterior wall of the ventricle toward the lower portion of the trabeculae. It has no ascending branch. A second branch surrounds the foot of the pillar, passing thence to the neighboring trabeculae. The third passes around the pillar in front and to the side, and is distributed beyond to the posterior trabeculae, where it gives off a posterior lateral and ascending division." Mönckeberg has never found the recurrent branch to the septum as figured by Tawara. The upper part of the septum, on its right side, contains no ramification from the right branch of the bundle of His. Contrary to the majority of the descriptions and diagrams, the area of distribution of the network of Purkinje on the internal surface of the right ventricle is limited above by a line passing through the base of the anterior pillar of the tricuspid. This area corresponds to the part of the ventricle occupied by anastomosing trabeculae, to the cavernous or spongy portion of the ventricle. There is only one prolongation above this level. Behind the pillar, toward the tricuspid orifice Mönckeberg found some fibers of Purkinje, almost up to the free border of the posterior flap of the tricuspid.

The distribution is almost exclusively limited to the spongy portion of the right ventricle, but does not correspond to the entire zone. It is an error to regard all the muscular bundles and bridges which intercross in this region as provided with Purkinje fibers.

It is less easy to trace the distribution of the network of Purkinje in the left ventricle than it is in the right, because the fibers of the network here have a strong resemblance to ordinary myocardial fibers. Moreover, the transition is made fiber by fiber and at different levels. To differentiate the fibers of Purkinje from the ordinary myocardial fibers Mönckeberg utilized the test for the presence of glycogen which substance is found solely in the former. He thus established the absence of glycogen-containing

fibers in the entire upper part of the septum and anterior lateral and posterior walls of the left ventricle. One half, or at least the upper third, of the pillars of the mitral valve is equally devoid of the Purkinje fibers. We may assume then that the zone of distribution of the network of Purkinje in the left ventricle is limited above by a line passing through the middle or junction of the middle and upper thirds of the pillars. This zone therefore corresponds to the spongy portion of the left ventricle, just as is the case on the right side.

I need not insist on the well known fact that the fibers of Purkinje divide, subdivide, or unite in nodes or networks, from which formations very complicated structures arise. The important fact that we must bear in mind is that we have to do with a network and not with individual fibers.

In its entirety the ventriculonector is related intimately by its extremities, above and below, with the auricular and ventricular myocardial network. The transition from one to the other of these structures is almost insensible, so that the measurements given for the ventriculonector are only approximate and those of its different portions are equally indefinite. In the sheep, for instance, a constriction is found separating the node of Tawara from the bundle of His, but this is not the case in the human heart. The bifurcation of the bundle of His does not always occur at the same place, and from the bifurcation to the network of Purkinje the distance varies with the size of the heart, and the same may be said of the dimensions of the network itself. The bundle of His also shows variations in width. Sometimes it is flattened from side to side, sometimes from above downward. Often a little before its bifurcation, the section becomes triangular.

The figures given below, which I have borrowed from Tandler,¹¹ are of value only as showing the relative size of the structures. The node of Tawara measures 6 mm. by 2 to 3 mm. the bundle of His has a length of 10 mm. (Tandler), of 11 mm. (Lydia de Witt), of 4 to 14 mm. (Gaetani), of 18 mm. (Retzer—who includes node and bundle). The width is 2.5 mm. (Retzer), 1.6 to 4.4 mm. (Lydia de Witt), 0.7 to 3.2 mm. (Gaetani). The thickness is given as 1.5 mm. (Retzer), 0.2 to 0.7 mm. (Gaetani). We need remember only that the ventriculonector is an extremely slender structure, that does not measure, when condensed in node or bundle, a thickness of more than 2 mm. in the part lodged in the septum. Beyond the latter the right branch is a thin thread of the thickness of 1 mm. while the left is a thin sheet lining the septal wall of the ventricle. As for the network of Purkinje which terminates it, its area of expansion corresponds to the spongy portion of the ventricles only.

We may suitably consider a little more in detail the relation of the ventriculonector to the myocardium, properly speaking.

The node of Tawara passes upward by insensible gradations into the auricular fibers. This passage takes place not only posteriorly and on the right side of the node, as described by Tawara, but all around its circumference, as shown by Mönckeberg, with the exception of the left side which is fused with the fibrous septum. At the node of Tawara, therefore, there is continuity with the auricular myocardium, almost exclusively the right; but this continuity is effected only by the intermediation of certain thin fibers. To quote Mönckeberg, "The union is accomplished by thin fibers of parallel course sometimes united in small bundles. These fibers pass into auricular fibers or are spread out in the form of a tuft and appear to insinuate themselves into the network (of the node). To regard these 'passage fibers,' which are similar in structure to those found in the posterior portion of the node of Tawara, as a special part of the auricle, appears to me to be unnecessary, the more so, as in man it is impossible to establish the exact line of demarcation in the node or in the auricular myocardium. In the latter we may still trace the fibers of this type way up between the separate portions of the network of the node until they are finally lost."

The relations between the ventriculonector and the ordinary myocardium differ at the level of the bundle of His and at that of its first branching. Throughout their long course these structures have no connection with the ventricular myocardium, the researches of Mönckeberg being very convincing on this point. The perfect isolation of this part of the ventriculonector does not, however, result from the presence of an enveloping sheath. This structure has been shown to exist in the sheep and the calf, by the injection of mercury, first done by Sappey, this anatomist, however, being under the impression that he had injected the lymphatic network. The demonstration has also been made by the injection of India ink. But this sheath is not present in the human heart.

It is only at their extremity, where the fibers assume the aspect of Purkinje fibers, that an imperceptible transition occurs from the network of Purkinje to that of the ventricular myocardium. We have specified above the zone of dispersion of the network of Purkinje and therefore the zone in which this passage occurs. If then the ventriculonector conducts "something" to the ventricular myocardium, it conducts it directly, and first to the base of the pillars and the apical region of the ventricles. The base of the ventricles is the continuation of the pillars and of the apex. Morphologically all these parts are so disposed that the network of Purkinje is continued in the myocardial network of the apical, spongy portion of the ventricles; while the myocardial apical network in turn is continued into the myocardial network of the smooth basal portion of the ventricles. Pace¹²

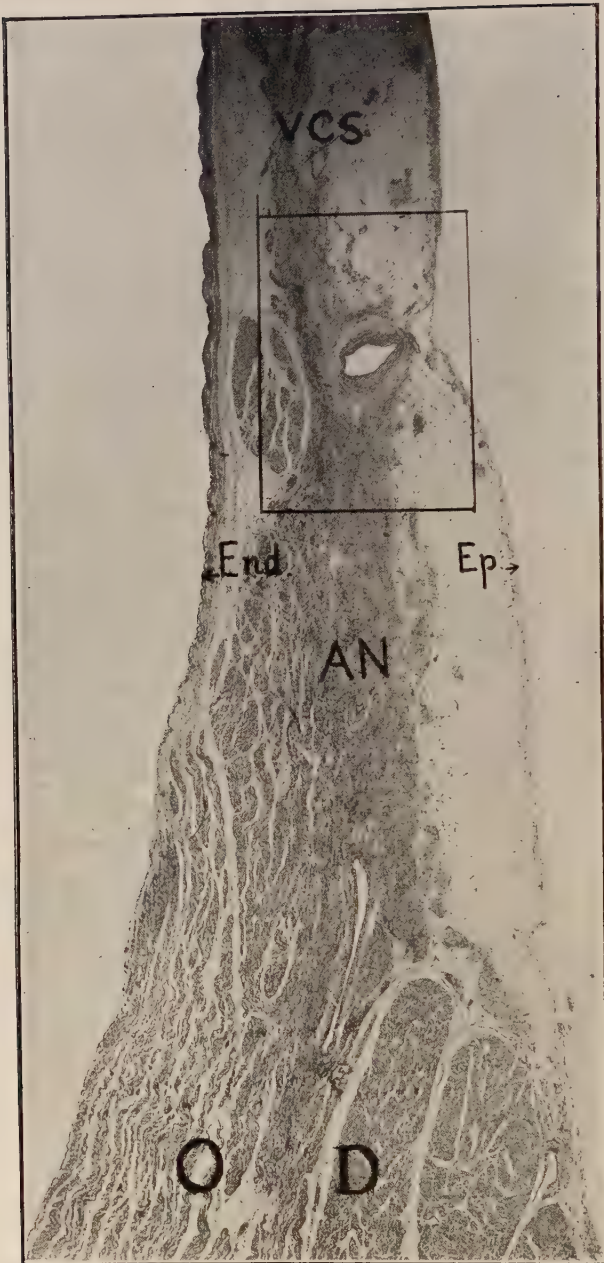


FIG. 27. The atrionector. At the junction of the vena cava superior, VCS, and the right auricle, OD, is the atrionector, AN, with its central artery cut nearly transversely above, and longitudinally below; End, endocardium of the auricle; Ep, epicardium and the subepicardial fatty layer.



FIG. 28. Part of the atrionector enclosed in a square in figure 27, shown under a high power. A, artery of the atrionector; NN, nerves in the substance of the atrionector; M, contractile auricular myocardium.

declares he has proved that the final ramifications of the bundle of His in the substance of the myocardium surround the adventitia of the great vessels; and he regrets that no one has found anything to confirm this statement. I have not been able to find this disposition of the fibers.

THE ATRIONECTOR

By this term we designate the structure or structures which are to the auricle what the ventriculonector is to the ventricle. The synthesis made by Tawara, who combined in one entity the atrioventricular node which bears his name, the bundle of His, and the network of Purkinje, has not been accomplished in the auricle, but analogous formations have been encountered, although so far they are still regarded as isolated structures.

Sino-auricular Node of Keith and Flack

Keith and Flack described in 1907 a myocardial structure comparable to the node of Tawara in formation. This is situated in the right auricle, close to the mouth of the superior vena cava. Since this discovery the endeavor has been made to establish the connection between this formation of Keith and Flack and the Tawara-His-Purkinje system, on the supposition that the former is the point of origin of the stimulus which initiates the cardiac contraction, the Tawara-His-Purkinje system being the conduction path of this stimulus to the ventricle. The research, however, has thus far been negative in its results, although it has demonstrated that the auricle, like the ventricle, possesses Purkinje fibers. The problem involved seems to us to be that of the connections of the node of Keith and Flack with the other elements of the auricle, the fibers of Purkinje and the myocardium. It is probable that these structures are susceptible of being united in a whole by a synthesis analogous to that established by Tawara for the ventricle, but for the moment we must limit ourselves to a description of the elements of such a synthesis which, while probable, is still hypothetical. These elements are the sinoauricular node of Keith and Flack and the auricular Purkinje fibers.

The term sinoauricular node of Keith and Flack is classically given to a mass of myocardial tissue situated at the point where the superior vena cava enters the right auricle. The venous portion of the node is situated along the sulcus terminalis, a groove which runs between the two caval veins and separates the portion of the auricle which proceeds from the venous sinus and atrial canal from the expansion of this canal where the pectinate muscles appear. The node of Keith and Flack lies between the muscular fibers of the superior vena cava, its tapering lower extremity being insinuated be-

tween the auricular myocardial fibers. These are disposed parallel to the fibers of the node and gradually become indistinguishable from them. Between the two extremities, inserted between the venous or auricular fibers and later confounded with them, the outer surface of the node is free where it is in contact with the subpericardial fatty tissue forming a yellow spot, visible at times on the heart; its deep surface is in close contact with the auricular tissue. By almost its entire surface therefore, the node is in connection with either the venous muscular tissue or the auricular myocardial tissue. We need not attribute any absolute value to the measurements given, but it is stated that in man the length is one centimeter and the breadth three to five millimeters.

Duplication or Duality of the Sino-auricular Node. Bruni¹³ has shown that in the ruminants there is found not only the formation just described situated to the right of the superior vena cava, but an analogous accessory formation to the left of this vein. Domenico Pace, confirming a statement which he had made in 1911, described in 1924 the existence in the lamb of a very peculiar disposition of the "specific" tissue of the auricle. This special muscular tissue forms a horse-shoe which embraces the bridge uniting the superior vena cava and the base of the auricle. The right posterior branch, which is the longer, is the node of Keith and Flack; the left anterior branch, shorter by half a centimeter, constitutes the new part, as yet unmentioned in the classical descriptions. Both parts are lodged in an adipose tissue, rich in fibers and nervous ganglia and each is accompanied by a satellite artery.

Pace¹⁴ notes that the new branch described by him rather tends to become subpericardial, while the branch which corresponds to the node of Keith and Flack burrows toward the endocardium.

Riccardo Segre¹⁵ in 1925 discovered an analogous disposition in man. He examined four human hearts sectioned in series, two being from the fetus, one from a newborn infant, and one from a young subject. He made a plastic reconstruction of one of the fetal hearts according to the method of Born, and found in addition to the node of Keith and Flack, a second formation of the same type, situated in the interauricular septum and extending to a point beneath the endocardium. The author was unable to decide whether or not the two were joined.

I have tried in vain to find this formation of Segre in the left auricle of the adult heart, but the agreement in the findings of Bruni, Pace, and Segre is not to be ignored, and will justify all the researches which others may make in the direction indicated.

Auricular Fibers of Purkinje

"According to all appearances," writes Tandler, "it was Thorel who was the first to describe the fibers of Purkinje in the auricle; before that, however, Schönberg had already been struck by the resemblance between the so-called bundle of Wenckebach and the cells of Purkinje." Thorel had described these fibers as forming a system of connection between the sinoauricular and atrioventricular nodes. This conception, combated especially by Koch and Mönckeberg, has now been abandoned. But the existence of the fibers of Purkinje in man has been confirmed by Freund, while Schwarz and Pace have found them in the lower animals. Tandler states that as long ago as 1905 he found in the heart of an executed man some fibers of Purkinje in the interauricular septum and the two venae cavae. Gibson, quoted by Lewis,¹⁶ described in 1909 fibers closely resembling the Purkinje fibers which he found in profusion in the auricle, more especially near the superior vena cava. Mönckeberg thought these were purely pathological formations, but Tandler objected that they had no resemblance to any known lesion of the myocardium, and that one could hardly imagine a pathological process which would modify the auricular myocardial network in well-localized and always identical regions.

The following is the description given by Tandler of the auricular fibers of Purkinje: These fibers are disposed in more or less important tracts which are of four types: (*a*) long tracts well individualized by a connective-tissue sheath from which are given off prolongations which subdivide the tract into distinct territories; (*b*) long tracts without any sheaths; (*c*) bundles of variable calibre but mixed more or less with auricular myocardial fibers; (*d*) bundles which are but slightly differentiated and hardly to be distinguished from auricular bundles. Types (*a*) and (*b*), better characterized, are found at the level of the venae cavae where they are disposed circularly; they are found also close to the sinoauricular node and in the zone of transition between the septal and posterior walls of the right auricle, especially near the opening of the coronary sinus. They are also to be found at the superior border of the right auricle where they bridge over the intraauricular groove in order to reach the left auricle. Finally they are to be found in the vicinity of the atrioventricular node. It must be noted that Tandler found these fibers only in the adult auricle. In the child, however, we find in the corresponding regions muscular fibers which differ in their greater calibre and greater richness in sarcoplasm from the neighboring auricular fibers.

The connection of the auricular fibers of Purkinje with the sinoauricular node has not been determined.

Those writers who make no mention of these fibers believe in a direct continuity between the network of the node of Keith and Flack and that of the auricle, the transition being insensible and embracing the entire circumference of the sinusal node. Those who admit the existence of auricular Purkinje fibers declare with Tandler that the fibers "undoubtedly penetrate into the sinusal node." But this anatomist acknowledges that he is unable to prove the existence of a union with the nodal network.

We should therefore hold in reserve this question as to the connections of the fibers of Purkinje with the auricular myocardial network. We have seen that the cells of the type *d* of Tandler are confounded with auricular fibers. We may assume then that there is an insensible transition at the boundaries of the two networks. A seductive hypothesis would be then that the sinoauricular node, dividing into two, is continued by a network of Purkinje extending to both auricles. The complete structure would be in a way to the auricle what the ventriculonector is to the ventricle. Let us add the possibility that a direct connection, without the intervention of Purkinje fibers may exist between the node of Keith and Flack and the auricular myocardium, just as one seems to exist between the node of Tawara and this same auricular myocardium.

STRUCTURE OF THE CARDIONECTORS

If one recalls the descriptions by the authors of the structure, in man, of all of the formations which we have assembled under the name of cardionectors, one will be struck by the fact that this structure is, in a general fashion, the same in all cases. Between the node of Tawara and that of Keith and Flack the difference described is of little significance; it consists mainly of a greater abundance of connective tissue in the former. Between the node of Tawara and the bundle of His the resemblance is almost perfect; the fasciculate aspect of the longitudinal fibers being only a deceptive one, for we have to do here always with a network made up of anastomosing tracts—a network more compact in the node; but broader, with more elongated meshes and richer in connective tissue in the bundle. The node of Tawara tends to assume a fasciculate aspect at its upper end which contrasts with the compact aspect of the network in the main part of the node. Tawara distinguishes in his description of the node a fasciculate and a reticulate portion. All of these formations may be summed up as constituted by a reticulate syncytium.

The muscular tracts which form the network may be resolved into fibers of quite regular contour although presenting sudden and frequent changes of calibre, sometimes veritable constrictions comparable to those described

by Ranvier in myelin fibers. Fine transverse or oblique septa divide them into imperfectly limited areas as one may observe in the contractile network of the myocardium. These areas correspond to the cells just as little as they do in the ordinary myocardium.

Under a high magnification each of these fibers appears to be rich in sarcoplasm within which are fibrils sometimes confined to the surface, sometimes scattered through its entire thickness. These fibrils pass from one fiber to another in the meshes of the network and it is their presence which gives to the fiber its longitudinal striation, which, however, is incomplete. Furthermore, there is, although not constantly, a transverse striation. Oval nuclei, vesicular or at times lobate, are found habitually in the substance of the fiber, surrounded apparently by a clear zone.

The meshes of the muscular syncytium are filled with connective tissue which is more abundant in the node of Keith and Flack and in the bundle of His than in the node of Tawara. Numerous elastic fibers are intermingled. The result of this is that the selective staining of elastic tissue, for example Weigert's, brings out a sufficiently marked contrast between the myocardium of the cardionectors and the adjacent ordinary myocardium to aid in the differentiation of the two.

The fibers of Purkinje, auricular and ventricular, have practically the same structure as the condensed portion of the cardionectors. They are perhaps richer in sarcoplasm and poorer in fibrils, whence they are paler, especially in sections stained with hematoxylin-eosin; also the transverse striation appears to be absent. They are wider than the fibers of the nuclei and of the bundle of His, but there are many variations. Finally they alone contain glycogen, which is the only shade of difference remarked between the fibers of Purkinje and the remainder of the cardionectors.

If we compare the syncytium of the cardionectors with the general myocardial syncytium, we see that the differences there also are minimal. In the zones of transition between the two syncytia it is really impossible to determine the boundary line between them, for we pass insensibly from one to the other, at least in the human heart, with which only we are dealing at the present time.

To quote Tandler: "Toward the extremities the branches of the bundle of His resemble increasingly in structure the musculature of the ventricle; and the two, at least in the human heart, are always most difficult of differentiation." Tawara wrote that "the last ramifications of the bundle of His become more and more rich in fibrils, the arrangement of which recalls that observed in the myocardial fibers; so much so that one can hardly definitely separate one from the other." He says further



FIG. 29

FIG. 29. The artery of the atrionector. Radiogram of a heart injected with a mass opaque to the x-rays. The artery of the atrionector, A.A.N., rises in this specimen from the right coronary, CD. CG is the left coronary. (From the Atlas of Jamn and Merkel.)



FIG. 30

FIG. 30. The artery of the atrionector. (Photograph of a dissection preparation.) View from above the dome of the auricle, showing the course of the artery of the atrionector (right anterior auricular type); the two ++ mark its origin from the right coronary, CD, and its termination at the atrionector at the level of the intercaval pole; VCS, vena cava superior; VCI, vena cava inferior; VP, VP pulmonary veins; AO, aorta; AP, pulmonary artery.

"One finds at times in the midst of the musculature of the ventricle isolated and scanty fibers resembling the fibers of Purkinje" (observations made on a subject who died cachectic).

Mönckeberg states that "The fibers coming from the left branch resemble more or less the ordinary myocardial fibers, and as a result they can be followed only with difficulty and at times this even becomes impossible." Further, in reference to the node of Keith and Flack the same author writes "It is impossible in man to distinguish exactly between the node and the auricular myocardium, the transition from one to the other being practically almost imperceptible.

To sum up: If one takes the morphological viewpoint, all of the syncytial network which goes to form the totality of the auricular and ventricular cardioneurons is obviously of uniform structure. Between these different parts there are only shades of difference, and moreover even these shades are inconstant. Furthermore, between the syncytial network of the cardioneurons and the myocardium proper the differences are not fundamental. They are absent where one network passes into the other. In the zone of transition it is usually impossible to distinguish the boundary line.

We can conclude, then, that the entire myocardium, the cardioneurons included, is uniform in structure. This is composed everywhere of syncytium, that is, a single mass of protoplasm, strewn with nuclei but without independent cellular territories. The syncytium is reticulate throughout. The only difference is in the disposition and the dimensions of the meshes of the reticulum; there are compact meshes in the nodes of Keith and Flack and of Tawara, and elongated ones in the bundle of His and its branches: more complex in the network of Purkinje, but elongated and again broader in the cardiac walls; very voluminous meshes containing not only connective tissue, but in addition lacunae filled with blood in the reticular tissues of the apex of the heart or of the auricles; finally giant meshes constituting the auricular and ventricular cavities.

All of the myocardial mass then may be considered as a sarcoplasmic mass in which contractile fibrils are differentiated. But this fibrillary differentiation is unequally distributed. In certain zones it is quite sketchy, in others it is very crowded so that, if we could suppress in thought the fibrils, we would obtain a sort of negative marked by sarcoplasm abundant in one place and scanty in another, yet all forming one continuous mass. Now, matters are so arranged in the heart that the regions with abundant sarcoplasm—the sarcomyocardial regions, one might say—control those poor in sarcoplasm—the contractile myocardial regions. In the ventricle

especially this disposition is evident; the sarcomyocardium controlling the myocardium of the region of the papillary muscles and of the cavernous tissue of the apex, and this myocardium in its turn controlling the myocardium of the base.

The sarcomyocardial regions then have nothing essentially specific except their position. In each cardiac cavity, auricle or ventricle, a sarcomyocardial region controls the contractile myocardial region—a sequential formation. In the auricle it is the atrionector which controls the auricular myocardium, and in the ventricle it is the ventriculonector which controls the ventricular myocardium. In each cavity sarcomyocardium and contractile myocardium pass insensibly one into the other. But it is through the sarcomyocardial region that the reticulate cardiac syncytium is in relation with the rest of the economy. The sarcomyocardial regions are in some sort the hiluses of the contractile myocardium of the auricular and ventricular cavities.

THE CIRCULATORY SYSTEM OF THE CARDIONECTORS

The capital rôle of the cardionectors in the cardiac mechanism is accentuated by the fact of a special circulatory arrangement.

*Artery of the Atrionector*¹⁷

Keith and Flack, in their description of the sinoauricular formation which goes by their name, point out that this structure is traversed by an artery situated at the junction of the sinus and the canalicular portion of the right auricle.

The section illustrated in Fig. 27 shows this artery divided, in the upper portion of the picture in a direction perpendicular to its course, and in the lower portion parallel to it. In this section the artery passes from the sinusal part of the node to the auricular part. But it may also be found passing in the opposite direction, from the tail to the head of the atrionector, a peculiarity which we shall understand better when we know its origin and course. The artery of the atrionector is remarkable for its large calibre. It is further distinguished by the considerable development of the muscular elements in its middle coat. From this it may be readily inferred that there are many possible variations in its delivery, under the action of its contractile muscular fibers.

I will add that the researches of B. S. and Adèle Oppenheimer¹⁸ have shown that there exists in the wall of the artery a plexus of fine nerve fibrils corresponding to the vasomotor type of Dogiel.¹⁹ The authors describe these fibrils in the pig, sheep, calf, and dog, but they have not been able to

detect them in man. Nevertheless, their existence is very probable and the difficulty of their demonstration is comparable to that encountered by Wilson²⁰ in connection with the nervous fibrils of the bundle of His in man, although this demonstration is easy in the case of the lower animals.

The Constant Presence of the Artery of the Atrionector. The atrionector is always centred by an artery. The source of this artery is variable but its termination is remarkably constant. All anatomists have emphasized this fact. Mouchet²¹ describes the intercaval region which corresponds exactly with the region of the atrionector, as a sort of "attraction pole" in which an auricular artery always terminates, either to the right or left, anterior or posterior. In more explicit fashion Gross²² insists on the existence of an artery in this region and names it from the fixity of its termination, the *ramus ostii cavae superioris*. Werner Spalteholz²³ described it under the name of *ramus cristae terminalis*, also calling it after the site of its termination. He has sought to give an explanation of the constant presence of an artery in this region and considers two hypotheses. He remarks first that the situation of an important vessel in the groove which separates the right auricle, properly speaking, from the intercaval region, might be explained by the fact that at the time of the contraction of the auricle, the flow of the blood in this artery would not be hindered. However, the course of the artery is not always parallel with the groove. The second hypothesis which he is more disposed to accept, is that this groove has a phylogenetic significance. In the reptile it lodges a well-developed artery, or sometimes two arteries faced end to end. From this fact he considers that the artery of the atrionector (*ramus cristae terminalis*) is phylogenetically a very old vessel and corresponds to this reptilian artery. I believe it is simpler to admit that the very important function of the atrionector, situated at this point, calls for a constant supply of arterial blood, hence the constant presence of an artery.

Abandoning this purely angiological viewpoint, and taking into account primarily the function of the region which the artery irrigates, it seems legitimate to name this vessel the artery of the atrionector. In the strict sense this artery is only a branch, terminal or collateral, which occupies the center of the sinoauricular node; but if we consider that this branch is far more important than the numerous muscular branches given off by the main trunk to the auricular wall, it seems to me proper to give, by extension, to the principal trunk the name which actually belongs only to the terminal portion. The name suggested by Crainicianu²⁴ of a nutrient artery of the node of Keith and Flack might otherwise be retained, but I prefer that of artery of the atrionector, as being shorter.

Drs. Mouchet and Nouredine²⁵ accept the name proposed by me of artery of the atrionector, but think it would be suitable to "reserve this term for the branch which enters the sinoauricular node rather than to apply it to the auricular artery which supplies this branch. The physiological importance of the territory irrigated by the artery of the atrionector should not prevail over the anatomical characters of the vessel studied, by giving its name to all of the arterial territory."

Origin of the Artery of the Atrionector. This artery, so constant in its termination, has a very variable origin. To obtain a clearer concept before examining this origin in detail, one might say in general, that the artery of the atrionector originates at some point in the incomplete circle formed, on the one hand by the right coronary, and on the other by the left coronary and its auriculoventricular branch. Occasionally the artery of the atrionector may not belong to the coronary system, but may be given off from an extracardiac artery.

Gross notes that the artery of the atrionector arose, in 60 per cent of the hearts examined by him, from the right coronary, and in 40 per cent from the left coronary. This origin always occurs at a slight distance, about 1 cm., from the origin of the coronary.

Mouchet has taken no account of specific formations in his description, otherwise so clear, of the arteries of the heart. Nevertheless, one may readily find in his description the artery which we are studying. It arises sometimes from the right, sometimes from the left coronary, anteriorly, at a short distance from its origin, or, as Gross failed to note, at the height of the left border. Mouchet also describes the following variation: the artery which terminates at the intercaval region, may arise, in 3 per cent of the cases, directly from the aorta. Its aortic orifice is near the orifice of the right coronary, from which it is separated only by a thin spur. Finally, W. Spalteholz who described the artery in question under the name of *ramus cristae terminalis*, notes that it occurs most frequently as an anterior auricular artery and more rarely as a right lateral auricular artery. He gives a series of figures representing the artery in the heart of the newborn, of the child, and of the adult. We see in these illustrations that the artery of the atrionector arises either from the right coronary, ten times as right anterior auricular artery (*ramus atrialis dexter anterior*), three times as right lateral auricular artery (*ramus atrialis dexter intermedius*), or from the left coronary, three times as left anterior auricular artery (*ramus atrialis sinister anterior*) and three times as left lateral auricular artery (*ramus atrialis sinister intermedius*). In one case (Case R), Spalteholz' illustration leaves us in doubt as to which of the anterior auricular arteries represented,

right and left, terminates in the atrionector. This case aside, we see that of the nineteen first cases the artery of the atrionector arises from the right coronary 13 times, and from the left coronary 6 times. The ratio given by W. Spalteholz, namely, 68 to 69 per cent from the right and 32 to 38 per cent from the left coronary, is little different from that of Gross (60 and 40 per cent respectively).

Before I was aware of the relatively recent work of Werner Spalteholz I had undertaken to verify the description of Gross and dissected the artery of the atrionector from its origin to its termination in eight hearts, and despite the small number of hearts studied, I had come to the conclusion that the variability of this artery was considerably greater than stated by Gross. Thus, of my eight cases the artery originated 4 times from the right coronary and 3 times from the left, while in the other case the origin, which I will describe, was quite exceptional.

Having dissected through their entire course the right and left coronaries and the auriculoventricular branch, I found no auricular artery ascending as far as the region of the atrionector. But on searching in this same region for the nutrient artery, I found it readily, and working backward from its termination, I was able to trace it toward its origin. The artery wound around the right pulmonary vein anteriorly, then curving upward passed between the two branches of the pulmonary artery, and turned toward the left. A snip of the scissors at the autopsy unfortunately made it impossible to determine the exact origin of the artery. This was either from the aorta below the last of the two great trunks which the arch gives off, or from a branch of the aorta. I could not determine this exactly, but what I could affirm positively was that this artery, so important in the physiology of the heart, did not originate from the coronaries, but was extracardiac.

I was interested therefore in the following finding in the work of Spalteholz. Working on the heart of a newborn, and on two adult hearts, this author found at the posterior-superior portion of the dome of the left auricle, in the vicinity of the pericardial reflexion, where the parietal becomes visceral, a good sized artery of extracardiac origin. Spalteholz, like myself, and for the same reason, was unable to determine the exact origin. This artery regularly gave off a branch to the interauricular septum. In one case the artery branched immediately after having penetrated the heart, and in the two other cases it furnished branches to the auricular dome and the diaphragmatic wall of the left auricle.

Finally Koch, quoted by Spalteholz, is said to have observed a similar fact in serial sections of a fetus of 6 to 7 months, and in this case the artery in question came from the left bronchial and was richly ensheathed by fas-

ciculi of the left pneumogastric. It terminated behind and to the left of the mouth of the superior vena cava.

Mouchet and Nouredine, having reviewed their radiographic documents and made new dissections in consequence of the article in which I had described this abnormal origin of the artery of the atrionector, declared themselves unable to establish its existence. They give as their opinion that "the pericardial artery of which Spalteholz speaks, runs most frequently from below upward in the pericardial leaflet, about 1 cm. from the cul-de-sac, upon the vertical intercaval space. This artery gives off an inferior branch which goes toward the inferior vena cava, to be distributed to the extra-pericardial portion of this vessel. It also gives off a superior branch which terminates at the point where the pericardium no longer covers the posterior aspect of the superior vena cava."

Neither Gross nor Spalteholz nor myself have found the artery described by Koch, namely, one given off from the right coronary, under the aspect of a posterior auricular artery, and ascending between the pectinate muscles to terminate in the region of the atrionector. It is this variation which Lewis notes (p. 21)—in the dog it is true, but from what we know of the variability of the origin of the artery of the atrionector in the human heart also, we ought to admit the possibility of the existence of this anomaly.

This long and detailed description of the origin of the artery of the atrionector has proved that there is good foundation for the general formula which I gave at the head of this chapter, namely, that the origin of the artery takes place at some point of the circle formed by the right coronary on the one hand, and by the left coronary and its auriculo-ventricular branch on the other. From some point of this "crown" there ascends toward the intercaval pole of Mouchet an artery destined for the atrionector, a sort of half-meridian, either as an anterior auricular artery, or as a lateral artery, or very likely also as a posterior artery (Koch's case). In a little more than half the cases, it is from the right semicircle, that is the right coronary, that this artery arises; in a little less than half the cases it arises from the left semicircle, and therefore from the left coronary or its auriculo-ventricular branch. Or finally, and this is the most curious anomaly, the artery of the atrionector arises not from a coronary, but from an extracardiac artery, such as the left bronchial.

From the variability of origin of this artery, we have to conclude that in a given case, where the clinical symptoms might lead us to specify the state of the circulation in the atrionector, nothing should lead us to prejudge the origin of this artery. In each individual case it would be necessary to trace the coronary artery from its aortic origin, first following the right and en-

deavoring to find the auricular branch at some distance from the aorta at a point in the midst of the adipose tissue where the coronary is buried. If we are unable to find the anterior auricular branch, or if, when found, it is soon lost in the anterior portion of the auricle, the best course is to follow the right coronary further, and we shall be able to locate the artery of the atrionector in the guise of a lateral or even a posterior artery. If the dissection of the right coronary has proved futile, the same research should be carried out with the left coronary or its auriculo-ventricular branch. If this last quest gives no result the artery in question must be sought along the line of reflexion of the pericardium in the hilar region of the heart. A more simple procedure, however, consists in exploring the region of the atrionector in the first place. We can find readily enough in the auricular wall an artery lodged in the sulcus terminalis, and in following it from its termination we can trace it to its origin.

Course of the Artery of the Atrionector. Between its variable origin from some section of the coronary circle, and its fixed termination in the intercaval region, there is a portion of the course of the atrionector artery which will vary according to its point of origin.

(1) The artery arises from the coronary, close to the aortic origin of the latter (anterior auricular type). The artery of the atrionector is given off about 7 or 8 mm. distant from the aortic origin of the right or left coronary. It runs behind toward the anterior wall of the right (or left) auricle, which bounds Theile's canal posteriorly. Running horizontally, the artery arrives in front of the interauricular septum, whence it passes upward in the anterior wall, more or less buried in the myocardial tissue. It skirts the auricular dome, along one of the sides, right or left, of the superior vena cava and reaches the sulcus terminalis, where it ramifies in the tissue of the sinoauricular node or atrionector. It may terminate there or give off a collateral branch to the atrionector and pursue its course for some distance beyond the atrionector. According to Gross, the artery may remain relatively superficial in its course or it may become deep. In this last case it is from the point where it reaches the interauricular septum that it buries itself in the latter; after having run a longer or shorter course it again becomes relatively superficial at the auricular dome, although always intraparietal.

(2) The artery is given off from a coronary artery when this vessel winds around the border of the heart (lateral auricular type). Figure 31, type 6, shows an artery arising from the auriculo-ventricular branch of the left coronary, when it winds around the left border of the heart and behaves like a lateral auricular artery. It ascends from left to right in the wall of

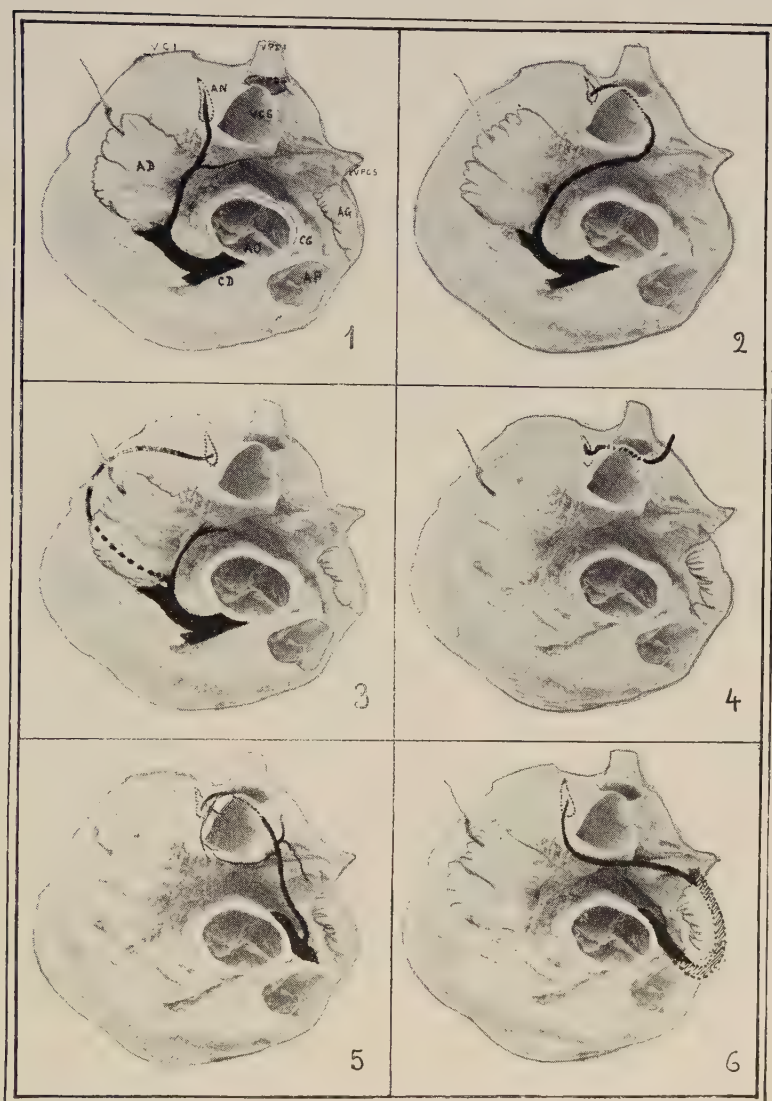


FIG. 31. Some variations in the origin and course of the atrionector. 1. Right anterior auricular type (see fig. 30). 2. Variant; the artery passes to the left of the vena cava superior. 3. Right anterolateral auricular type. 4. Extracardiac-origin type; the artery comes probably from the left bronchial artery. 5. Left anterior auricular type. 6. Left lateral auricular type.

the auricle, reaches the auricular dome, and winds around the superior vena cava in front and on the right side. A variation of this type, which might be designated the anterolateral auricular type is sometimes found. Here the artery arises from the right coronary about 2 cm. from the aortic origin of this vessel. It gives off an ascending branch which passes upward in the anterior auricular wall, but faces out before it reaches the superior vena cava. The principal trunk curves on itself toward the right and follows a course parallel with that of the right coronary. Behind the base of the right auricle, the artery of the atrionector suddenly turns upward and gains the auricular dome and the region of the atrionector, conducting itself like a right lateral auricular artery.

In the lateral auricular type, we may note the following curious disposition of the artery. At the base of the auricle the artery makes a sort of hernia into the cavity of the latter, being covered only by a very thin layer of myocardium. This disposition bears quite a close resemblance to that of a pulmonary arteriole in a tuberculous cavity in the initial phase of Rasmussen's aneurysm.

(3) The artery arises from the coronary circle at the posterior margin of the border of the heart. This variety is probably that described by Koch, and its course as well as its origin has already been described.

(4) The artery has an extracardiac origin. The course of such a variety varies evidently according to its origin. An idea of such a course may be obtained from the description of one such anomaly described by me above.

Distribution of the Artery of the Atrionector. The arterial branch which irrigates the atrionector is, as we have seen, only one of the divisions of the auricular artery from which it is given off. This main artery gives origin in its course to several collaterals, the description of which we take from Mouchet: "(1) A branch which is distributed to the adipose tissue forming a pad on the surface of the aorta. (2) Branches which go to the right auricle. (3) Branches which lose themselves in the anterior surface of the auricle. (4) The arteries of the interauricular septum. (5) The right and left auricular branches."

The main artery, according to Mouchet, most frequently divides into two terminal branches, one antero-superior and one postero-inferior. "The anterior branch, often thinner than the other, branches out into terminals at the mouth of the superior vena cava and in the space which separates the vena cava from the upper edge of the base of the auricle. It often forms a loop which curves around the orifice of the superior vena cava. Sometimes also this loop is formed by two terminal branches of the vessel. Of these the posterior, generally the more developed, forms on the superior

aspect of the dome a sort of arcade, which terminates between the two venae cavae, in the region which one of us has termed the intercaval pole, at the level of the right superior pulmonary artery. It is this latter branch which must be regarded as the artery of the atrionector in the strict sense of the word."

I do not believe that it is necessary to be quite so dogmatic as Mouchet, but I agree with him that the arrangement which he describes is of frequent occurrence. His opinion merits the more consideration because it rests upon the study of a large number of hearts. There are still other arrangements of the artery that may be observed; thus, if we study the atlas of Jamin and Merkel²⁶ we see that the main artery may terminate in a very varied fashion. I believe, however, that we should reserve decision regarding the termination of the artery of the atrionector. The question is not yet definitely settled, and the researches of Bruni, Pace, and Segre seem to show that there may be a doubling of the sinoauricular node. If this particular configuration of the node is confirmed, it will be necessary to concede to the other branches—in particular to that, which according to Pace supplies the left half of the node—the same physiological rôle as that of the artery of the atrionector in the strict sense of the term.

Anastomoses of the Artery of the Atrionector. The arterial circle of Keith and Flack is described by these authors in their memoir as a sort of arterial collar which assures the special circulation of the sinoauricular node. This arterial collar surrounds the opening into the auricle of the superior vena cava. It is formed by the anastomosis of three arteries: (1) the anterior auricular derived from the left coronary; (2) the anterior auricular from the right coronary, and (3) the lateral auricular, also from the right coronary.

Koch agrees with Keith and Flack as regards the presence of a pericaval arterial circle, but Koch declares that this is formed of branches given off from the right coronary only.

According to Gross the ramus ostii cavæ superioris (that is to say the artery of the atrionector) arises from one or the other of the coronaries but never from both together. It may, however, form the pericaval circle by means of its anastomotic branches.

Werner Spalteholz, finally, concludes that the arterial circle is present but once in three times. In seven cases studied by him it was formed three times by a left anterior auricular artery, once by a left lateral auricular artery, twice by a right lateral auricular artery, and once only the arterial circle was formed by vessels proceeding from both coronaries, the right furnishing the chief supply. This last case only is comparable with

that described by Keith and Flack. The Keith and Flack collar, formed by the anastomosis of several auricular arteries, is then an exception. Moreover, in a general way Spalteholz insists on the fact that the right auricle, and more especially the intercaval region, is rarely the seat of arterial anastomoses, although it is richly irrigated. This is not the case in the left auricle where anastomoses are frequent. Moreover, as Mouchet has observed, an artery may not be terminal anatomically and yet be so in the physiological sense. In other words it may show actual anastomoses developed by artificial injection, but these will not play any rôle in the functions of the living heart.

The question which is raised here belongs really to the domain of anatomo-clinical methods. If we consider the functional troubles which the atrionector frequently presents, and which probably are of circulatory origin, we may well believe that the special artery of the atrionector is a terminal artery, and that no other can perform its office. It may best be compared to one of the arteries of the encephalon.

Artery of the Ventriculonector²⁷

The ventriculonector is essentially an intraseptal formation and one need not feel astonished that its nutrient artery is a septal artery. In reality it is the highest of the posterior septal arteries.

Under the name of septal (or perforating) arteries we include a double series of arteries which are given off—the anterior from the anterior descending interventricular branch of the left coronary; the posterior from the posterior descending interventricular branch given off as a rule from the right coronary. They penetrate into the septum and pass off two by two in opposite directions. Figures 32 and 33, taken from the atlas of Jamin and Merkel, show these septal arteries.

The artery of the ventriculonector is the uppermost of the posterior septal arteries. This essential character of the septal artery justifies the name under which it is described by Haas²⁸ and adopted also by Gross, viz., "*arteria (vel ramus) septi fibrosi.*" But the fact that it is from this artery that the branches destined for so important a function as the supply of the ventriculonector arise would seem to justify me in giving it the name of artery of the ventriculonector. With this introduction I will proceed to a brief description of the origin, course, and termination of the artery in question.

Origin. Being the highest of the posterior septal arteries, the artery of the ventriculonector takes its origin from the coronary which terminates at the upper part of the posterior interventricular groove. Here the groove

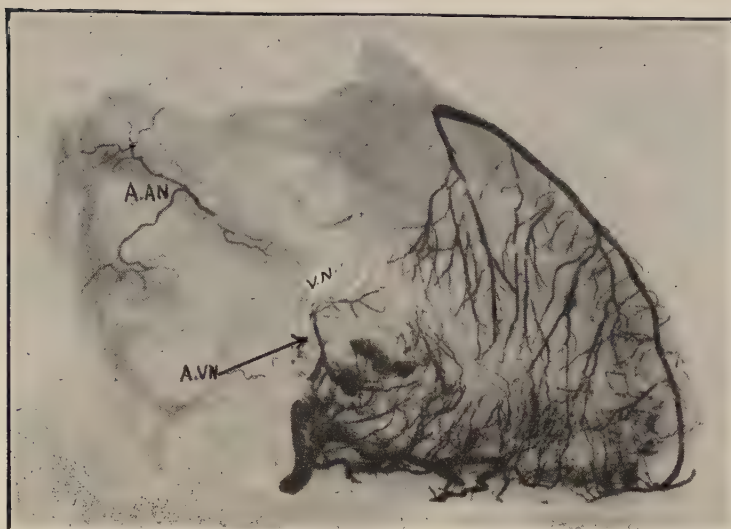


FIG. 32. The artery of the ventriculonector. Radiogram of a heart injected with a mass opaque to the x-rays, showing the double series of anterior septal (above) and posterior septal (below) arteries. The arrow points to the artery of the ventriculonector, the highest of the posterior septal arteries, giving branches to the ventriculonector, represented schematically at VN. A.AN, artery of the atrionector (cf. fig. 29).

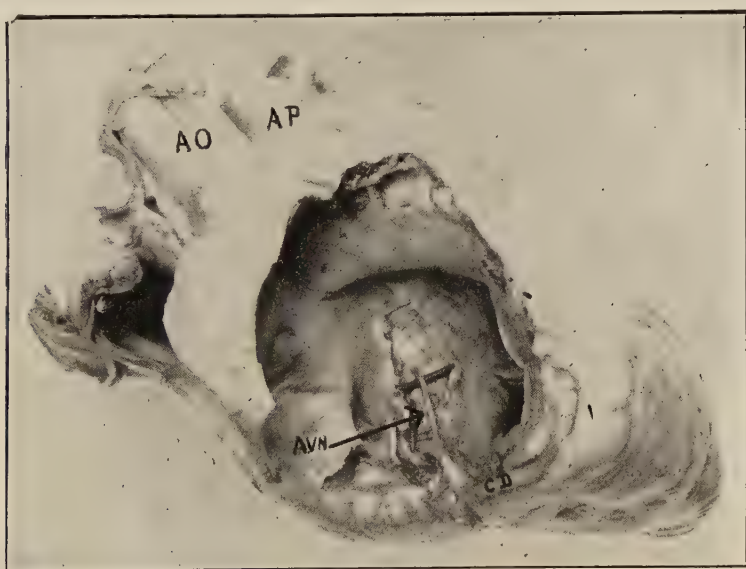


FIG. 33. The artery of the ventriculonector. (Photograph of a dissection preparation from a subject aged 20 years.) The septal segment of the tricuspid valve was cut away and then the endocardial and auriculo-ventricular myocardial layers were removed. Thus the artery of the ventriculonector, AVN, is exposed; a straw is passed under it just where it enters the fibrous septum. CD, right coronary artery; AO, aorta; AP, pulmonary artery.

which separates the two hearts is crossed by the groove which separates the auricles from the ventricles. This point of crossing is, according to the terminology of foreign authors the "crux" or "cross" [so named by Gross].

According to the findings of Gross, in 86 per cent of all cases it is the right coronary which terminates at the cross and in only 4 per cent is the left coronary concerned; in 10 per cent both reach the cross, but in the latter case it is the right coronary which in six cases out of ten gives origin to the artery of the ventriculonector. Hence in 92 per cent of all cases the artery of the ventriculonector arises from the right, and in only 8 per cent from the left coronary.

Haas concluded from his researches that the artery of the ventriculonector is always derived from the right coronary. My own researches, although they are based on a relatively small number of cases, have shown that in two cases out of eight the artery arose from the left coronary, and W. Spalteholz has made similar observations. The conclusions of Haas are therefore somewhat too arbitrary.

I would add, with Gross, that there has never been a case in which more than one coronary, right or left, has given origin to the artery in question, and with Haas that this special artery is never absent.

Course. The artery, supplied ordinarily by the right coronary, is given off from the latter just in front of the cross. The right coronary bends inward soon after its origin and becomes the posterior interventricular artery, supplying throughout the entire length of the groove ten or eleven posterior septal arteries which penetrate into the septum from behind forward.* The artery of the ventriculonector, which represents the uppermost of these posterior septal arteries, passes beneath the terminal portion of the coronary sinus and running from behind forward, rests first in the connective tissue layer which cleaves the interauricular septum, then penetrates the interventricular fibrous septum. It is an artery of appreciable calibre, easy to find and to follow by dissection from its coronary origin to its entrance into the fibrous septum.

In the young subject its course is nearly rectilinear and its thin walls with scanty musculature, contrast with the thick and muscular walls of the main

* G. Haas says that the second and third posterior septal arteries (the artery of the ventriculonector being the first) do not, like the subsequent ones, originate in the posterior ventricular artery, but are supplied by a common trunk which is given off from the right coronary above the artery of the ventriculonector. This trunk is termed by him the "ramus superior ventriculorum." This disposition, considered by Haas as constant, does not exist in the two hearts, one of which is represented in Fig. 33. The arrangement is that which we have shown,

coronary. In the elderly atheromatous subject, on the contrary, its walls are thick and its course spiral. When the artery of the ventriculonector is given off from the left coronary the latter passes by the lower surface of the venous coronary sinus and bifurcates at the cross. One branch descends into the posterior interventricular groove, giving off posterior septal branches, while the other, the highest of these septal branches, penetrates between the two muscular layers of the auricle and ultimately follows the same course as when it is given off from the right coronary.

In its course through the interauricular septum the artery of the ventriculonector reaches the head of the imaginary man (node of Tawara), and passes directly through it. In its course through the interventricular fibrous septum it passes through the body of the man (bundle of His), not through the middle but excentrically. The artery lies beneath the body of the man, between it and the crest of the muscular wall on which it rests. A perpendicular section brings this arrangement well into view. Beyond the bundle of His the artery gains the muscular septum properly speaking, where it ramifies to a greater or less distance.

Branches of Distribution. In its quality as septal artery, the artery of the ventriculonector supplies the muscular septum by means of arterial branches which we will rapidly sketch. I have just spoken of its terminal branches. Among the collaterals may be mentioned, on the right side, branches which ascend on the right side of the interauricular septum, and branches passing down beneath the median or septal leaf of the tricuspid valve, supplying the superficial layers of the right side of the interventricular septum. On the left side, a branch (*a*) ascends vertically in the fibrous septum, then abruptly curves toward the base and a little forward, and runs upon the left side of the interventricular muscular septum. Another branch (*b*) is directed toward the posterior part of this septum. Finally, a third branch (*c*) pierces the fibrous septum, ramifies again on the left side of the interventricular septum, and is distributed to the initial portion of the left branch of the bundle of His.

The branches which interest us most, however, are those destined for the ventriculonector. They are given off from the artery of the ventriculonector or from one of its septal collaterals which we have just mentioned, and in particular branch (*a*). It is, however, impossible to give a description which will hold good for all cases. The greatest variations occur and each should be studied individually.

The following is the description of Haas which corresponds closely to that of Gross: "The artery of the fibrous septum (artery of the ventriculonector) reaches the node of Tawara at its posterior-inferior extremity. It sends a

finely subdivided branch to the latter (the "ramus nodi atrioventricularis" of Haas). It passes through the node of Tawara from behind forward and continues into the bundle of His. At the anterior extremity of the bundle of His it subdivides into fine ramifications which terminate soon after entering the two branches of this bundle. At times the artery terminates in the node of Tawara without continuation into the bundle of His. The latter is then supplied by a collateral given off above the node of Tawara."

Our own findings are in accord with the preceding descriptions. In hearts studied in serial sections I have seen the artery of the ventriculonector give off a posterior branch for the node of Tawara and the initial portion of the bundle of His; that is to say for the base of the ventriculonector. It may also give off an anterior branch for the terminal portion of the bundle of His and the initial portion of its branches of bifurcation, that is to say, for the apex of the ventriculonector.

But the point on which insistence should be laid, in my opinion, is the variability of ramification of the arteries of the heart in general. Hence the conclusion that in the case of the ventriculonector, as in that of the atrionector, we should regard the indications given above as possessing only a general orientation value. One can draw no reliable anatomo-clinical conclusion from a single observation. It is necessary to make a complete study of the heart in question as to the circulation of its cardionectors, beginning with the origin in the coronaries and following the vessels to the termination of the branches destined for the cardionector supposedly injured.

The Accessory Arterial Branches of the Ventriculonector

The artery of the ventriculonector, just studied, supplies the greater portion of the ventriculonector (node of Tawara, bundle of His, and the initial portion of its bifurcating branches). The remainder of the ventriculonector (terminal portion of the branches of the bundle of His and the network of Purkinje) receives arterial branches from a different source; or it may also possibly receive its nutriment from the artery which we have described; that at least is my impression. I believe that it is especially the adjacent contractile myocardium and not the sarcomyocardium that is supplied by the arterial branches ordinarily supposed to supply branches of the bundle of His. We will now consider these accessory branches.

The Right Branch of the Bundle of His. This branch, as we know, follows the trabecula marginalis or ansiform band. In this muscular band there is always found a branch of an anterior septal artery, that is to say, an artery given off from the left coronary. This is the ramus limbi dextri of Gross. Mouchet describes it under the name of artery of the anterior

column of the right ventricle. This author says: "By its constancy, its course, and its special distribution, this artery merits a more detailed description. Given off from the second perforating (anterior septal) artery* a few millimeters from its origin, the artery of the anterior column of the right ventricle passes into the ansiform band which it follows for its entire length to a point at the base of the column. It divides in the substance of this structure into a certain number of branches which continue on to its termination. Its length is from 35 to 40 mm. In its totality it describes a long curve, the concavity of which is directed upward and to the right. It is often divided before its entry into the ansiform band, and we then find two arteries running into this myocardial column parallel with its long axis. More rarely the two arteries are given off separately from the anterior perforating artery." At the extremity of the anterior wall of the right ventricle where the band is prolonged into the anterior column, jutting into the cavity of the ventricle, other arterial branches, given off from anterior and descending branches of the right coronary, penetrate into the band and anastomose with the previously described branch, which itself comes from the left coronary.

Whether these arterial branches which are regarded as assuring the nutrition of the right branch of the bundle of His are destined to it in part, or whether they supply only the contractile myocardial tissue properly speaking is something which it is impossible to decide definitely.

Left Branch of the Bundle of His. This ramus spreads out into branches at the internal surface of the left ventricle, the ramifications being grouped in an anterior and a posterior bundle. We have already seen that one branch (*c*), given off from the artery of the ventriculonector, is distributed to the region where the initial portion of the left branch of the bundle of His is situated. Both the anterior and the posterior bundles are said to receive their arterial blood supply through numerous anastomoses between the anterior and the posterior septal arteries, of which the anterior come from the left coronary, and the posterior habitually from the right coronary. Consequently, the anterior bundle obtains its blood especially from the left coronary, the posterior bundle from the right coronary. We must, however, make the same reservation here as we did in respect to the right branch.

Network of Purkinje. This network, formed from the terminal filaments of the two branches of the bundle of His, does not appear to have an autonomous circulation. We may ask ourselves, "Does it participate in the

* Mouchet has occasionally seen it originate from the third, and only in very exceptional cases from the first, perforating artery.

circulation of the adjacent layers of the myocardium against which it rests, or is it nourished by the same artery as the rest of the ventriculonector?" It is difficult to decide.

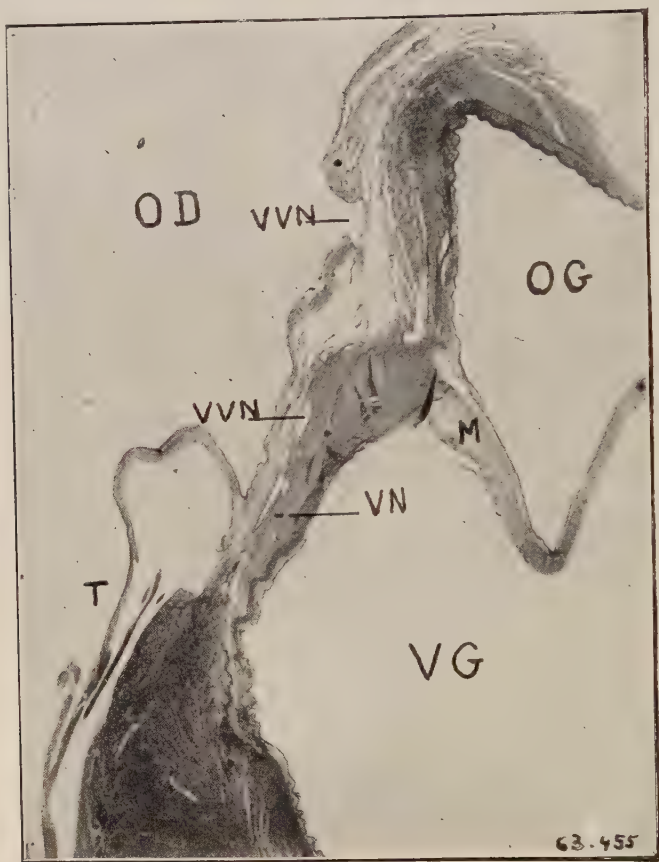


FIG. 34. The vein of the ventriculonector. The ventriculonector, VN, is seen cut horizontally in the septum between the two auricles—right, OD, and left, OG. VG, left ventricle; M, mitral valve; T, tricuspid valve. The vein of the ventriculonector cut in the ventriculonector and at the point of its opening into the right auricle, VVN, VVN. Cf. this section 455 with sections 420 and 520, shown in figures 17 and 18, which precede and follow it, in which we find again the vein V, V.

Anastomoses. The question of arterial anastomoses in the ventriculonector calls for the same observations as those made in the case of the atrionector. Gross holds that the branches destined for the bundle of His, and supplied habitually by the right coronary, anastomose at their anterior

extremity with the corresponding branches of the uppermost of the anterior septal arteries which are branches of the left coronary. Haas, on the contrary, asserts that there are no anastomoses between the territories of the right and left coronaries in the atrioventricular node and bundle of His.

S. Spalteholz says that at times anastomoses between the branches of the anterior and posterior septal arteries may be seen on the left wall of the septum, immediately behind the left branch of the bundle of His. But he adds, on the other hand, that a minute examination shows, in the upper part of the septum, branches which seem to press each other aside, the anterior leaning to the left, the posterior to the right. Finally, he is unable to decide as to the presence or absence of anastomoses in the bundle of His itself. When a preparation has been well injected it is precisely then that it gives an image which is indecipherable, because too rich.

Moreover, the value of anastomotic connection as regards the blood supply of a part is not a question of anatomy to be resolved by such a coarse method, for instance, as injections. There is, as Mouchet expresses it, a veritable "threshold of anastomotic sufficiency" which experimentation might perhaps determine. But in this case the findings would hold good only for the animals studied.

Upon the whole it is the anatomo-clinical method which will throw most light upon the question put here, as to the reality of physiological anastomoses in the ventriculonector. Certain observations made by Mönckeberg weigh in favor of the absence of efficient anastomoses; he has found alterations in the ventriculonector while the adjacent myocardial tissue remains sound, and *vice versa*. (See cases XXIV, XXV, L, LVIII, LIX.) The anatomo-pathological findings of Mönckeberg show, besides, that even if the fibers of the ventriculonector represent an autonomous arterial territory, this territory may include the nearest of the adjacent myocardial layers. But we shall have to look to new researches for removing the ambiguities on these points.

Venous Circulation of the Cardionectors

The *ventriculonector* is drained by an important vein (*the vein of the ventriculonector*) which empties into the right auricle about 2 mm. in front of the free border of the valve of Thebesius. The venous orifice is recognized by the naked eye, and seen upon the right face of the interauricular septum. I think it is always present. It is one of three foramina described by Lannelongue.²⁹ The vein belongs to the group of the *venae minimae Thebesii*.

The vein of the ventriculonector is readily demonstrable in the bundle of

His where it accompanies the artery of the ventriculonector closely, and, although in separate channels, vein and artery both perforate the fibrous septum. The vein then empties at the level of the node of Tawara, into a larger vein which comes from the interventricular muscular septum. Thus enlarged by the vein of the ventriculonector, it pierces the node of Tawara and then passes into the connective-adipose tissue interposed between the myocardial layers of the right auricle and the left ventricle. Here the vein leaves the artery, the latter dipping toward the base to rejoin the coronary which gave it its origin. The vein ascends and then forms a right angle terminating at the venous orifice which we have described.

I have not observed any valves in the course of the vein. In one case, on studying serial sections, I noted that this vein was very sinuous above the cross of the septum. I thought at first that there was a double pair of valves, but this appearance was due to the flexuous course of the vein at this point. The venous blood of the ventriculonector passes through a clear channel into the right auricle.

The word "vein" as used here, is hardly justified, for the structure of this vessel is extremely simple. The wall consists solely of an endothelial layer covering a thin connective tissue layer, and there is no muscular tunic. Near its termination the auricular bundles run parallel to the venous canal but have no connection with it; in particular there is no formation which resembles a sphincter.

The Venous Circulation of the Atrionector. My conclusions regarding the veins of the atrionector will be less definite. I have not found here a satellite vein analogous to that accompanying the powerful artery which centers the node of Keith and Flack, but only several small venules, especially recognizable on the side of the endocardium where the node is covered by the auricular myocardium. It has seemed to me that these venules empty into the cavities of the cavernous tissue situated on the internal aspect of the crista terminalis, therefore, into the sinusal part of the auricle. The complexity of the meshes of this tissue makes it difficult to reconstitute with certainty the ultimate course of the venules of the atrionector. All that can be affirmed with certainty is that in carefully following the auricular wall on the side of the septum, no trace of any venous orifice has ever been found. It seems probable, therefore, that it is in that part of the atrium situated more to the right, to the inner side of the crista terminalis, that the venous blood of the atrionector empties into the right auricle.

From this study we may conclude that the two cardionectors have a venous return circulation through the veins which all terminate in the right auricle. The veins of the atrionector empty into the upper part of the

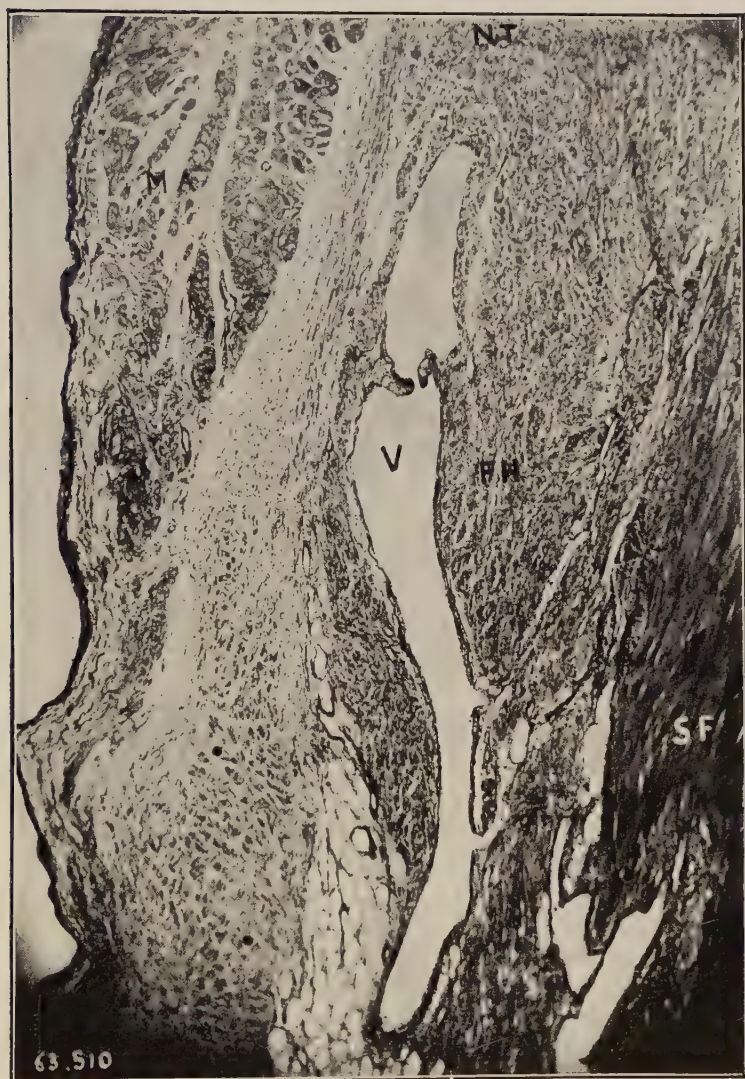


FIG. 35. The vein of the ventriculonector. Section 510 highly magnified, compare with section 520 (fig. 18) under lower power. V, vein of the ventriculonector; NT, node of Tawara; FH, bundle of His; SF, fibrous septum; MA, myocardium of the auricle. The appearance of a valve in the vein is an artefact caused by an infolding of the wall of the vessel.

sinusal portion of the auricle, into the vestibule of Keith; while those of the ventriculonector discharge into the inferior portion of the auricle, into the atrial canal close to the ventricular border.

The Lymphatics of the Cardionectors

I have only to open and close this chapter for I have no personal data to supply on the subject, nor have I found in the classical descriptions anything which treats with this question. Mönckeberg limits himself to the following statement: "In the connective tissue (lodged in the meshes of the muscular network of the bundle of His) which, especially in front, envelops the bundle on its lower surface, there are often lymphatic apertures which are remarkably wide, as we have also noted in the fetal heart." The relatively recent and very detailed monograph of Otto C. Aagaard³⁰ does not mention lymphatics associated with the cardionectors. He has shown very clearly that it is not the lymphatics which are injected at the level of the expansions of the left branch of the bundle of His, in the heart of certain animals, but their sheath. The latter, however, does not exist in man.

GANGLIA AND NERVES OF THE CARDIONECTORS

This section should be one of the most important, for it is in reality the whole subject of the ganglia and nerves which should be treated here, but that is a question the elucidation of which calls for long research. It is easy to establish the presence of nerve ganglia, especially in the vicinity of the sinoauricular node and the interauricular septum and close to the mouth of the coronary sinus, where there is a veritable "nervous constellation" following the expression of Pace³¹ who gives a good description of it. In the sinoauricular node one can easily recognize the nerve fibers and ganglia. Wilson has described, although only in certain animals, multipolar ganglionic cells in the bundle of His.

What is most necessary for us to know, however, is the connection of the nervous elements, ganglia and nerves, with the nervous system, especially the pneumogastric and sympathetic, on the one hand and with the myocardium on the other. This is a domain of wide extent which has been only partially explored. See more particularly articles by Woolard and Blackhall-Morrison.³²

TECHNIQUE OF EXAMINATION OF THE CARDIONECTORS

The following is the technique which we have employed in our researches.³³ In order to make a profitable study of the cardionectors, it is advisable first to fix the entire heart without opening it, at the autopsy table. In

this way the relation of the different parts of the organ are preserved and the marking out of a block for removal is easy. Moreover, if it is necessary, as is often the case, to follow up any structure that extends beyond the limits of the first block, the removal of a second block is easy and the matching of the two series of sections obtained is assured, although at one part or another of the line of transition certain sections may be slightly displaced, but not so as to interfere with a good observation. Another point to remember is that the heart should always be fixed in the same way. The ideal procedure would be to fix the heart *in situ* by the injection of formol into the cadaver, although this is not indispensable. Indeed I have obtained entirely satisfactory results with hearts fixed after removal from the thoracic cavity. The vascular pedicles, especially the venous, should be divided as far as possible from the heart. The organ is then suspended in a vessel filled with the fixative liquid. A pin, bent in the shape of a hook, is fixed firmly in the wall of the aorta, and a thread, fastened to the pin, is attached at the other end to the stopper of the vessel. The cavities of the heart are filled with the hardening fluid, and the organ takes and maintains a shape very near the normal. Dr. Bordet and I³⁴ have been able to satisfy ourselves of this by comparing the measurements made on a heart thus hardened with those made on the orthoradiographic tracing taken from the same subject shortly before death. The hardening fluid, penetrating readily into the cavities, assures fixation, and also permits of good staining. After two or three days it is a good practice to open the cardiac cavities to remove any blood clots found therein which if left behind, would discolor the tissues and interfere with satisfactory fixation. After this lapse of time also the heart is sufficiently hard to enable one to remove a fragment. But it is better to wait a day or two longer, after cleansing the cavities. The hardening fluid employed is a 40 per cent aqueous solution of formaldehyde, called formalin, and of this a dilution is made of 10 parts to 90 parts of water.

Atrionector

We have habitually made sagittal sections, parallel to the long axis. An incision half a centimeter from each side of the intercaval groove delimits a band of auricular wall which contains the atrionector. It seems to us as if it would be well hereafter to carry the incision on the left side beyond the interauricular septum, being guided by the data furnished by Bruni, Pace, and Segre. The crista terminalis, a buttress of muscular tissue, which extends from before backwards, between the smooth sinusal portion of the auricle and the part rendered irregular by the cavernous tissue of the base of the auricle and the pectinate muscles of the auricular appendix, always



FIG. 36

FIG. 36. The opened right chambers of the heart, showing the atrionector, AN, the ventriculonector, VN, with its different parts—the node of Tawara, NT, and the bundle of His, FH, with its right and left branches, BD and BG (somewhat schematic). The line AB marks the section that must be made in order to get the part for blocking which includes the ventriculonector in its thickest portion. VCS, VCI, vena cava superior and inferior respectively, AO, aorta giving off the right coronary artery, the section of which is seen immediately above the line AB; this artery is divided again at the posterior border of the heart and is seen above the same line AB. Ap, pulmonary artery. SC, the coronary sinus. TR, the septal segment of the tricuspid valve.

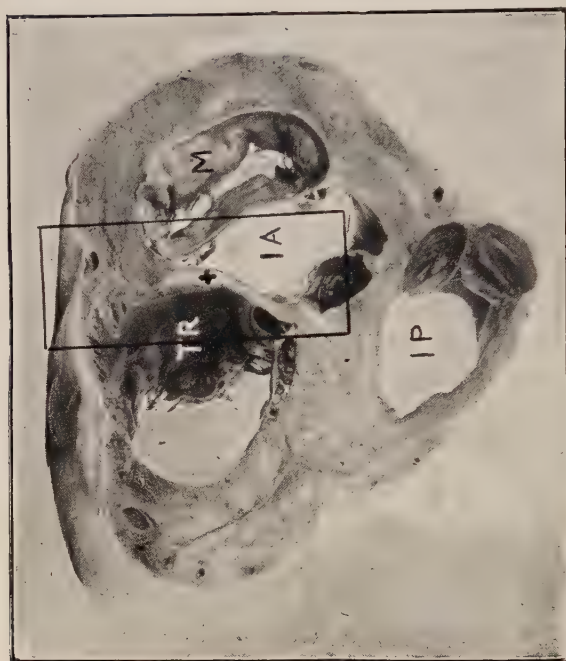


FIG. 37

FIG. 37. Surface exposed by the section through the line AB in figure 36 seen from above. IA, aortic infundibulum; IP, pulmonary infundibulum. TR, tricuspid valve; M, mitral valve. Septum between the right and left chambers of the heart. The rectangle bounds the part to be removed for blocking in order to study the ventriculonector and its artery.

encloses the atrionector. In front the block to remove should include a part of the superior vena cava, for the length of about a centimeter. A transverse incision, made 3 cm. behind the first, will liberate the block.

Ventriculonector

The technique for removing the principal portion of the ventriculonector is given here. This portion comprises the node of Tawara and the bundle of His with the initial portion of its branches. If it is desired to proceed beyond the branches and *a fortiori* to study the network of Purkinje, we must remove other blocks, keeping track in each case of the problem to be solved. In such a case it will be a matter of delicate research, the difficulty of which has been emphasized repeatedly by Tawara and Mönckeberg in their monographs.

It is in the right heart that it is easiest to obtain the inclusive block for examination of the bundle of His. The classical procedure for opening the right heart should be modified, for it mutilates the region of the atrionector. The following is our manner of procedure. The antero-external wall of the right ventricle, toward the infundibulum of the pulmonary artery, presents a depressible zone deprived of sub-pericardial fat. Above this fleshy zone is a layer of fat which masks the myocardium; it is the sub-sigmoidian region of the pulmonary artery. Below this fleshy zone the examining finger feels a resistance from the presence of the anterior papillary muscle of the tricuspid and the muscular tracts which unite it to the ventricular walls. At the level of the above mentioned zone which is deprived of fat and depressible, we make with scissors a window in the ventricular wall. This window is at first small, but gradually enlarged by successive removals of tissue. In carrying out this procedure we must make sure that we do not injure either the anterior papillary muscle, which may be inserted rather high up on the wall, or the septo-marginal tract or ansiform band, which terminates here. Through the widened opening we remove the layer of fibrin which is always found between the wall and the valve, and is prolonged into the pulmonary artery. The tricuspid valve and all of the pulmonary infundibulum are now clearly visible. After this we must remove most of the fibrinous clot which is often present in the tricuspid orifice and prolonged into the right auricle. The following incisions can now be made: (1) A long knife with a narrow blade is passed into the pulmonary artery (care being taken not to injure the semilunar valve), and emerges through a transverse incision at a point where the vessel is usually cut across, that is 2 to 3 cm. above the valve. We then incise the anterior wall of the right ventricle, as close as possible to the septum and arterial

wall. (2) The blade is introduced into the tricuspid orifice and passed into the right auricular appendix, the point emerging in the auricle, at a distance from the atrionector. We have thus a double V-shaped incision with the point directed downward which isolates a flap from the external wall of the right heart. (3) A third incision along the base permits the removal of the flap and exposes the cavities of the right side. To make this incision we raise the flap, seize it by its apex, and cut the base of the triangle, directing the knife so that it just grazes the aortic prominence. From before backwards this section includes the muscular arch beneath the aortic prominence, the antero-external leaflet of the tricuspid valve, and finally, the right auricle in its portion. Between the subaortic arch and the auricle the knife has passed through the solid fatty tissue and divided the right coronary artery.

The right aspect of the septum between the two hearts is thus well exposed, and we now proceed to determine the marks which enable us to circumscribe the block containing the ventriculonector. The great or septal leaflet of the tricuspid marks the limit between auricle and ventricle. The first landmark to be noted is in that region where the septal and the antero-external leaflets converge upon the septum, remaining for the most part adherent to the resected flap. Where these leaflets join, the septum which they cover is membranous—the *pars membranacea septi*. On the left side this same portion of the septum constitutes the *spatium intervalvulare* of Spalteholz, which is a triangle bounded by the two aortic semilunar segments, the right anterior coronary, and the posterior non-coronary, semilunar segments. One may also take for the first landmark a point situated about 1 cm. below the aortic origin of the right coronary. It is easy to note the site of this origin exactly for the section of the coronary is quite visible. By estimating at what distance from the aorta the section passes and taking into account the obliquity of the coronary at its origin, we can readily locate the point when the artery is given off.

The second landmark is the valve of Eustachius, stretching between the orifices, lying above the inferior vena cava and below the coronary sinus. About 2 mm. below the line uniting these two landmarks lies the bundle of His. Following this line we cut the septum, dividing it as nearly perpendicularly as possible. A good orientation of the block is thus obtained, and it remains to isolate the block while giving it the desired dimensions. A second section is made parallel to the one above noted, starting at a point a little below the level of the coronary sinus, which leaves the bundle of His above it. Behind we may cut immediately in front of the valve of Thebesius; and anteriorly beyond the small column or columns where the tendons begin which run to the most anterior part of the septal leaflet of the tricuspid valve.

The block thus delimited contains the node of Tawara and all of the bundle of His with its two branches in the first part of their course. The average measurements are 4 cm. in length and 1 cm. in each of the other dimensions. It may then be imbedded as a whole and sectioned without any interruption by means of any of the microtomes in current use. This holds good, however, only when making the sections horizontally, which is the preferred method. Frontal sections can be obtained only when the block is subdivided into two or three parts of suitable thickness. We then risk breaking the continuity of the series in consequence of a slight displacement of the section.

In the horizontal serial sections of this muscular block which has been removed in following our landmarks, the bundle of His appears as soon as the razor has passed the adherent inferior border of the anterior coronary semilunar segment. On the left aspect of the septum the insertion of the large mitral segment is seen, and on the right aspect the insertion of the antero-external and septal leaflets of the tricuspid valve can be noted.

It is between the mitral insertion on one hand, and the point of convergence of the lines of insertion of the septal and antero-external leaflets on the others, we find that the bundle of His extends. The septum here is half tendinous and half muscular. The tendinous portion corresponds to the origin of the aorta, blended behind and to the left with the large mitral segment. The muscular portion, behind the angle of the antero-external septal junction, consists of auricular fibers, especially those lined with the endothelium of the coronary sinus. In front of the angle of junction the fibers of the right ventricle, or infundibular fibers, taper in the opposite direction to some distance from this angle. Thus the muscular lining of the tendinous portion of the septum, relatively thick behind and in front, becomes thinner toward the middle and is wholly lacking at the junction of the lines of insertion of the septal and antero-external segments. It is the region corresponding to the spatium intervalvulare of Spalteholz on the left; and on the right to the tendinous triangle concealed by the segments of the tricuspid valve.

All of our sections measure $7\ \mu$ in thickness. As a rule we begin by picking up one section in every twenty, which enables us to note the details of the case studied. Next we mount all of the intervening sections necessary for the solution of the problem given. These sections are stained with hematin and the Van Gieson mixture, after previous passage through Weigert's fuchsin, the selective stain for elastic tissue. This staining darkens the cardioneurotensors, contrasting with the lighter tint of the myocardium where the elastic element is more scanty. We also use Mallory's mix-

ture. The contrast is very marked between the red muscular tissues and the blue connective tissue.

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CHAPTER III

PHYSIOLOGICAL HYPOTHESES

The problem of the mechanism of the heart is a complex one, and many hypotheses have been proposed for its elucidation. They should answer the three following questions: Under what influence does the contraction of the myocardium occur? Why does the heart contract rhythmically? How is coördination of the contractions of the auricles and ventricles effected?

WHAT CAUSES THE CONTRACTION OF THE MYOCARDIUM?

The myocardium may be made to contract artificially by causing an electric or mechanical shock to act upon any point whatever of its surface. But what is the normal stimulus?

The majority of authors hold that the myocardium contains in itself the cause of the contraction; these are the myogenists. Others consider that the myocardium contracts under the action of the nervous system; these are the neurogenists.

Automatism of the Myocardium

The myogenists believe that the myocardium is endowed with automatism. As a consequence of the discovery of the specific myocardium, it is more particularly to the latter that the property of automatism is attributed. In whatever part of this myocardium a stimulus might arise it would extend to the entire heart. But, as a rule, one special part is believed to be the seat of origin of the stimulus.

Hypothesis of a Major Center and Minor Auxiliary Centers. The usual place where the excitation arises is the sino-auricular node and this major center dominates the entire heart. English authors, particularly Lewis, term it the "pace-maker." The stimulus originating in the sino-auricular node, advances into the non-specific auricular tissue, like a ripple started by a stone thrown into the water, and determines the contraction of the auricles. Then the wave, reaching the boundary line of the auricle and ventricle is concentrated at the node of Tawara, and having passed down through the specific myocardium—the bundle of His and network of Purkinje—arrives at the contractile myocardium of the ventricles, causing its contraction. Lewis holds that the excitation wave has a speed of one meter

a second in the auricle, two meters a second in the specific myocardium, and fifty centimeters a second in the contractile myocardium of the ventricles.

According to this hypothesis the automatism of the specific myocardium is limited to the sino-auricular node, the rest of the myocardium, both specific and contractile, functioning only as a conductor of the impulse created in the major center. But under certain abnormal conditions it may happen that one major center ceases to be efficient or limits its action solely to the auricle. In such a contingency certain portions of the specific myocardium are seen to be minor auxiliary centers; the impulse originating there is seen to affect parts of the myocardium no longer influenced by the major center.

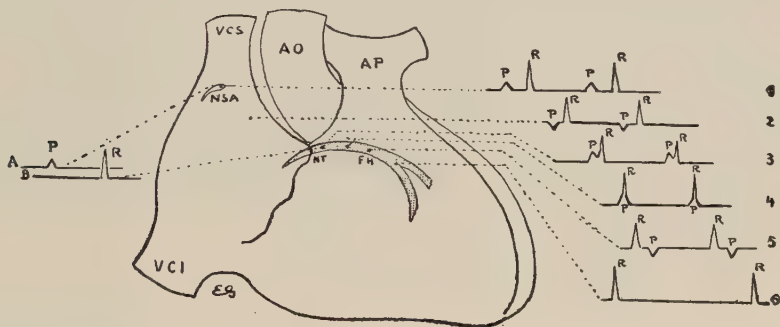


FIG. 38. Schematic representation of the sinus node and the minor centers with the types of rhythm, cardiographically expressed, which they control. The heart is seen on its right surface. VCS, VCI, superior and inferior venae cavae; AO, aorta; AP, pulmonary artery; NSA, sino-auricular node; NT, Tawara's node; FH, bundle of His. 1, The normal, or sinus, rhythm; 2, auricular rhythm; 3, 4, 5, the atrioventricular or nodal or septal rhythm, with its three varieties—supranodal, nodal, and infranodal; 6, the idio-ventricular rhythm. At the left of the figure are shown the two rhythms, one dependent on the atrionector, NSA, the other on the ventriculonector, NT-FH. The varying disassociation of the series of auricular P waves and of ventricular R waves gives all the combinations.

The existence of the following minor auxiliary centers (see fig. 38) is assumed:

- (1) Auricular centers in the auricular wall, without definite locations.
- (2) Atrioventricular centers, situated in the node of Tawara and therefore called nodal centers. These are also known as septal centers because the node of Tawara is a septal formation. According to their location—high, medium, or low—they are called supranodal, nodal, and infranodal centers.
- (3) A Hisian center, located in the bundle of His above its bifurcation. This is also known as the idioventricular center because its domain is limited to the ventricles alone.

To the major center and the minor centers correspond those rhythms of

the heart called sino-auricular or sinus, juxta-sinusal, auricular, atrioventricular (nodal or septal, with its three varieties: supranodal, nodal, and infra-nodal), and the idioventricular rhythm.

It is believed that of two centers the highest causes the more rapid rhythm, and that the more rapid center dominates the slower one. This explains why, so long as the sino-auricular node functions in its integrity, its rhythm is that of the heart as a whole. When, on the other hand, the ventricle is removed from the influence of the sino-auricular node the frequency of its contractions is not over 30, this figure being that of the rhythm of the minor idioventricular center.

Hypothesis of Two Major Centers. Professor Vaquez and Dr. Donzelot¹ maintain the view that the automatism of the specific myocardium is exerted always at two major centers, as well for the normal as for the abnormal heart. The first of these is the sinus center, the domain of which is restricted to the two auricles. The second is the atrioventricular center (nodal or septal), node of Tawara in the strict sense, which holds both of the ventricles under its dependence, by intermediation of the bundle of His regarded as a conductor. It would be consistent then to differentiate an auricular and a ventricular rhythm in the heart.

The Myocardium Contracts Under the Action of a Nervous Stimulus

The neurogenic theory at the present time has but few partisans. We have adopted it provisionally as a working hypothesis. According to this hypothesis the excitation is derived from the nervous ganglia of the heart—the autonomic system.

The capital objection to every neurogenic theory is based on the observation made by G. Fano. This physiologist saw the cardiac tube of the embryo of a chick commence to beat on the third day of incubation, when it contained as yet no nerve elements. Even if it is true that the heart beats before any differentiation of nervous elements, perceptible to our present means of investigation, it still may be unwarranted to conclude therefrom that, when this differentiation is made, the intrinsic nervous system of the heart has no part in its contraction. We believe that at a given time when a morphological differentiation is produced, there is developed also a correlative functional differentiation. We might mention the neuromuscular cells as an example. We are forced to accord to them the two functions of excitation and contraction; but then when the nervous cell and the muscular cell become differentiated each of them assumes its definite function. It is not unlikely that the elements in the vascular tube behave in the same manner. On the other hand, if the nervous cells do not

appear in the human heart until toward the fifth week (His Jr.), the immigration of the ganglion cells into the heart precedes by little the development of the sinusal node. In proportion as the nervous apparatus is completed the sinusal node reaches full development; at $4\frac{1}{2}$ months in the human fetus both are completely formed (L. Stiénon). The objection made to the nervous theory should then also be made to all theories which hold that the specific myocardium has an important rôle in the cardiac mechanism.

The scanty knowledge which we possess regarding the nervous system of the heart makes it prudent for us not to reject absolutely the hypothesis of an intervention of the autonomous nervous system in the contraction of the myocardium.

Why Is The Contraction of the Myocardium Rhythmic?

The law of periodic inexcitability, placed well in evidence for the heart by Marey, and for the brain by Richet and Broca, seems sufficient to account for the rhythm. Athanasiu,² however, has sought another explanation than the law of periodic inexcitability for the rhythmicity of the myocardium. He suggests that this is the result of the interplay of the primary neurons and the neurons of inhibition. Unfortunately he does not support this hypothesis by any precise anatomical data.

Demoor and Rijlant³ have sought the solution of this problem in quite a different direction. Their experience leads them to think that the rhythmic contraction of the myocardium is under the control of an internal secretion, a cardiac hormone. The following is one of their experiments: A fragment of the left auricular wall, separated from the specific myocardium, of a dog or a rabbit is the seat of isolated muscular twitches. But if we add to glucosed and oxygenated Ringer-Locke solution, a certain amount of a maceration fluid in which the node of Keith and Flack has been soaked, after a period of latency, about one hour, this fragment shows spontaneously rhythmic contractions. If the maceration liquid is heated to $63^{\circ}\text{C}.$, however, this phenomenon does not occur. The same result is obtained with a fragment of ventricular wall freed from endocardium and from the subjacent layer of specific myocardium (Purkinje fibers).

Demoor and Rijlant hold that the sino-auricular node is the principal seat of the secretion of the hormone. Haberlandt⁴ at about the same time and independently of these authors, made analogous experiments with cold-blooded animals, such as the frog, and showed that in two-thirds of the cases the addition to the Ringer's solution of an extract of the sino-auricular node, initiated, strengthened, and accelerated the heart beats. Contrary to the

results announced by Demoor and Rijlant, heating did not destroy the properties of the cardiac hormone. Moreover, Haberlandt maintains that the rhythmicity of the beats does not depend on hormone action but solely on the refractory phase of the myocardium.

It is extremely probable that humoral influence has a relationship to cardiac contraction. In this connection it may be stated that the presence of calcium and potassium in the blood is of direct importance. Calcium augments, and potassium diminishes the rhythmicity of the heart. In a medium deprived of calcium the heart ceases to beat. But it would seem that it is solely in the general fact that after a phase of activity a phase of rest occurs that we have to look for the cause of the cardiac rhythm. As for the action of initiating, strengthening, and accelerating the heart beats, noted by Haberlandt, an objection may be stated, which is that extracts of organs may have this triple action without a specific action being necessarily involved; adrenalin, for example may act as a "cardiac hormone."

How Is the Coördination of Contractions of the Auricle and Ventricle Effected?

According to the most popular theory, the fact that the wave of sinusal excitation reaches the auricle first, and the ventricle next, necessarily proves that the auricular contraction precedes and that the ventricular follows. The course followed by the impulse accounts for the coördination and the succession. The interval between the two contractions indicates the time occupied in the passage of the impulse from the auricle to the ventricle.

But in contravention of this theory we may recall the very exact experiments of Athanasiu who has demonstrated that the contraction of all of the parts of the auricle and even of both auricles occurs at the same instant; likewise there is a synchronism of contraction of the base and of the apex of one ventricle or of both. This synchronism excludes the hypothesis of a wave which has an appreciable velocity.

Drs. Vaquez and Donzelot, in order to explain the auriculo-ventricular coördination, bring in the play of the intracavitary pressures. It is the "stroke of the auricular pump" which determines the contraction of the ventricle. But it may be objected that this explanation is of no value in the case of an inverted sequence or of a heart which contracts while empty. The same objection holds good for a similar explanation proposed by Professor Bard.⁵

Demoor also seeks an answer to the question with which we are now concerned. "Why," he writes, "and how, can a wave of activity run through the heart regularly in bringing about the revolution?" He attributes it to

a diffusion of activity of the node of Keith and Flack transmitted by the bundle of His, and also to the continuity of the endosubendocardial plane (network of Purkinje) extending from auricles to ventricles. According to Demoor, "there exists then in the texture of the right auricle and ventricle, a continuous system suitable for the conduction in all directions of the consequences of any change whatever of the humors produced through the influences of the stimuli, and for the elaboration of the "active substances" which humorally give to the contractile fibers their special property of excitability and their capacity to contract rhythmically. This apparatus is represented by the nodal system (specific myocardium) of which the ventricular and auricular portions are, as we have demonstrated, connected by the atrioventricular bundle and the endosubendocardiac system." "The Hisian route represents a connection of rare perfection, destined to ensure a rapid, exact, and general appropriation of the humoral medium to the needs of the powerfully energetic action of the myocardium."

One may make to this explanation, which substitutes a humoral wave for the wave of excitation of Lewis, the same objection which we made with Athanasiu to the hypothesis of the wave of excitation. How explain that there is a synchronism in action of the different portions of the auricles and ventricles? One may again object that it is far from well understood why it is, in cases of discordance of auricular and ventricular rhythms, that the auricular and ventricular rates often differ. It would not be very satisfactory to reply that the activity of the "substance" of the specific myocardium is less in the ventricle than it is in the auricle.

Athanasiu endeavors to explain the coördination of the auricle and ventricle by the intervention of the nervous system. According to him, a co-ordinating nerve current of excitation leaves the sino-auricular node, gains the ganglion of this region and from thence the interauricular ganglia. It passes next to the node of Tawara, runs through the bundle of His and reaches the columnæ carneæ and wall of the ventricle. There would then be in some sort a "superior command represented by the nodes of Keith and Flack and of Tawara and the bundle of His." Although Athanasiu may not be very explicit on this point, it appears from his memoir that when he speaks of the nodes and of the bundle he has in view the nervous elements contained therein. This hypothesis, however, lacks an anatomical basis.

L. Frédéricq says:⁶ "The pulsation of the heart is a wave of contraction which begins in the right auricle, extends rapidly to the walls of the two auricles, then slowly crosses the bundle of His to irradiate rapidly in the substance of the ventricles. The synchronism of the two auricles is the necessary consequence of the anatomical continuity of the musculature of

the auricles and of the propagation to the left auricle, by muscular conduction, of the wave of excitation born in the right auricle in the node of Keith and Flack." But, in the opinion of this physiologist, the wave of contraction may originate at any point whatever in its muscular path. He states that "the initiation of the movement does not always take place in the node of Keith and Flack." And as proof of this, he alleges the fact that the suppression of the node of Keith and Flack determines neither a temporary arrest of the heart, nor inversion of the sequence PR, nor substitution for the normal rhythm of the rhythm called nodal.⁷ As Frédéricq recalls, Erlanger and Bachmann, Jager, Flack, Moorhouse, Magnus, and Alsleben had already noted this fact. But, on the other hand, it is true that Langendorff, Lohmann, Hering, Cohn, Kessel, and Mason have concluded from their experiments that extirpation of the node of Keith causes an arrest (at least temporary) of the heart, and in all cases a profound modification of the cardiac rhythm (of coördination), notably the appearance of the nodal rhythm (suppression of P) or of the coronary rhythm (shortening of the PR interval) or even of an inverted rhythm (RP).

Eyster and Meck and Demoor and Rijlant have more recently studied the rôle of the node of Keith and Flack in the initiation of the cardiac pulsation. In the majority of their experiments, these authors observed immediately after the operation a slowing of the rhythm and the disappearance of P. Then the wave P reappeared more or less quickly and the rhythm became a coronary one with a shortened PR interval. This rhythm was established immediately in the latest experiments of Rijlant. But L. Frédéricq has quite recently repeated his experiments and concludes that extirpation of the node of Keith and Flack gives insignificant results or none at all. "It does not notably alter the rhythm of the pulsations in the heart of the dog." But one may ask whether these divergences in the experimental results do not depend upon our ignorance, until recently, of the doubling of the sino-auricular node, such as is described by Bruni, Pace, and Segre. Frédéricq notes that when he extended the cauterization to the greater part of the wall of the right auricle, contiguous to the septum, he saw the nodal rhythm appear.

The problem is further complicated if one takes into account the result of the experiments of Rijlant in grafting the node of Keith. Rijlant succeeded in grafting the node of Keith by implanting the graft into the auricle of an animal previously submitted to resection, irrespective of whether the two animals were of the same or different species (goat and sheep). This implantation practised immediately after obtaining of the graft caused a return of the heart to its sinusal rhythm at the end of a few hours or in some

cases immediately. As the graft had lost its anatomical connections it is a question whether or not it "took" and became continuous with the surrounding tissues. Whether or not it acted through the intermediation of "active substances" taking the humoral route, as Demoor supposes, is far from clear.

Such are the principal responses which have been made to the different questions relative to the cardiac mechanism. I will set forth, as briefly as possible, the hypothesis to which I have been led.

Hypothesis of a Variable Circulation in the Cardionectors

Let us refer to the anatomical data set forth in the preceding chapter. We saw by them that the heart should be considered as constituted essentially by the local evolution of the primitive vascular tube, consisting in the development of a double annex—an auricular pouch serving as a reservoir of blood, and also as a propulsive chamber, and a ventricular pouch, serving only as a propulsive chamber. Those annexes developed by the cardiac tube are dominated by what I have proposed to term cardionectors. Each of these annexes possesses a cardionector; the atrionector controls the auricle; the ventriculonector rules the ventricle. The functioning of each of these cardiac pouches is subordinated to the corresponding cardionector. What does the cardionector transmit to the contractile myocardium? We are still on unknown territory and the debate between the neurogenists and the myogenists is not settled.

The myogenists, and in the front rank Frédéricq, claim the existence of a "cardiac pulsation" originating in the myocardium and transmitted by it. This conception gives a good enough account of the auriculo-ventricular sequence and in general of the mechanism of the normal heart. The physiologists may be satisfied with it. But clinicians know from practical experience the frequency with which, in the abnormal heart, the auricle and ventricle beat with a different rhythm; and it becomes difficult to understand how a pulsation arising up-stream can give rise to auricular and ventricular pulsations of differing rates. Or one may imagine that something in some way has "minted" the original pulsation, recoinning it into secondary pulsations, varying in number for each of the two chambers.

Hypothesis for hypothesis, we can more readily entertain an action of the nervous system, and a fact established by Athanasiu seems to us suggestive in this connection. In his experiments with direct electrocardiography, Athanasiu registered certain electric undulations which he thinks indicate a nervous influence. The rhythm of these waves is about 600 per minute. That is precisely the maximal rhythm at which the auricle works in auricular

tachycardia. It seems to us probable then that each cardionector struck by the waves with this rate of 600, permits, or not, the passage of this permanent undulatory current and at a fixed rhythm. It acts like a connector; whence the name for it proposed by us—cardionector.

L. Stiénon has been led by his researches on the sinusal node to a conception very near our own. He concludes thus: "An impression is gained that the sinusal node, from the anatomical point of view, has not the appearance of a generator of rhythm, but rather that of a relay, placed between the auricular myocardium and the intracardiac nervous apparatus,—a sort of protective mechanism against the influences which emanate from the ganglia and which are transmitted to the cardiac muscular fibers."

It is possible that the conductivity of the cardionectors depends on their chronaxia. We will recall, with Vaquez and Donzelot,⁸ in this connection, that Lapicque and his collaborators, Mlle. Veil and Frédéricq,⁹ have shown that the excitability of the ventricular muscle and that of the ventriculonector (bundle of His) are in a fixed relation of 1 to 3 and that when this relation varies to an appreciable degree the stimulus is no longer transmitted to the ventricle. There transpires here a phenomenon analogous to that demonstrated by M. and Mme. Lapicque,¹⁰ in their initial researches on the motor nerve—skeletal muscle system. The notion of chronaxia, which we owe to these authors, will emphasize further the phenomenon on which we insist—the variability of the conductivity of the cardionector.

The conductors, being independent, may function differently and allow the impulse going to the auricle to pass in a different way from that going to the ventricle. The discordances of rhythm would be interpreted as differences of activity of the cardionectors. So long as the functioning of the two cardionectors is well-balanced the auricular and ventricular rates are normal and equal. But if this function is disturbed there is anisorhythmia or discordance.

Among the factors capable of influencing the function of the cardionectors, one of the most important seems to us to be the circulatory system, and as each cardionector has its own circulation, it can be readily understood that the activity of one alone may be affected, or that both of them may be equally modified. I have brought to the support of this theory of the cardionectors with variable circulation positive anatomo-clinical facts, which will be found set forth in this book. It is therefore in a disturbance of the circulation that we must seek the immediate cause of the anomalies of the cardiac mechanism which we propose to pass in review. It is necessary to remark that from now on we allude only to the human heart, which possesses a coronary system. The frog, so often employed in these experiments, does not possess this structure.

The circulatory system of the cardioneotors depends then on two factors, the entry of arterial blood on the one hand and the exit of venous blood on the other. Modification of the arterial supply itself depends on the action of the left ventricle which propels the blood into the coronary system. To this ventricular action there should probably be added the action of the muscular coats of the artery, more especially because the coronary arteries and those of the cardioneotors have a well developed muscular tunic. A word should be said as to the action of this tunic. Its exact rôle has not yet been explained, but we may nevertheless from now on conceive that the contraction of the muscular coat of the artery influences the circulatory motion in two opposite ways, according as it is tonic or clonic. A tonic contraction of sufficient duration will diminish the delivery of blood by the artery, while conversely, a series of clonic contractions will augment it.

Without doubt also the capillary system influences the circulation, but its functioning is still little known. In the phenomena classed habitually under the terms vasoconstriction and vasodilatation, a function is imputed to the capillary system which really probably belongs to the arterial. We need only examine by the capillaroscope the vascular loops in the skin of the tips of the fingers, at the nail-beds, or, by the corneal microscope, the vessels which mark the transition between arteries and veins, to be convinced that between the arterial and venous systems there is no capillary network, but only very short vascular segments, non-muscular connecting links, which are incapable of regulating the circulation of the parenchyma of the tissues in which they are included, by either their contraction or their dilatation.*

The influence of the venous outflow on the circulatory system should not be underestimated, however, because it is an important anatomical fact that the venous emissaries of the cardioneotors always empty into the right auricle,—those from the atrioneotor in the upper part, from the ventriculoneotor in the lower part. The cardiac mechanism is thus closely related to both the arterial and the venous circulation. According to the case the cardioneotor will be brought into contact with a fluid medium, both the quality and quantity of which may vary, depending upon its renewal. No wonder then that the activity of the neotor will vary in proportion to differences in the medium in which it lies.

Nevertheless, one difficulty persists. How does this hypothesis explain the auriculoventricular coördination? I have proposed the following ex-

* It is possible that the caliber of the connecting vessels may be modified in accordance with variations of volume of the endothelial elements, determined in turn by humoral differences.¹¹

planation. Of the two arteries of the cardionectors, whatever their origin, that of the atrionector is shorter than that of the ventriculonector. Taking as a starting point a contraction of the left ventricle, the time which will elapse up to the moment when the ventricular propulsion is first evident in the cardionectors will be less for the atrionector than for the ventriculonector. The first will reach its flood and go into action before the second; and as a result the contraction of the auricle will precede that of the ventricle. The PR interval is an indication of the time elapsing between the arrivals of the two waves.

It is well understood that it is not the ventricular wave which elicits the following auricular contraction, for in cases in which there is a discordant ventricular bradycardia, we find that two, three, or more contractions of the auricle occur after a single ventricular contraction. The ventricular contraction does not, therefore, call forth, in sequence, the contractions first of the auricle and then of the ventricle, the latter giving rise in its turn to the succeeding auricular and ventricular contractions. But the wave of blood may influence the rhythmic function (the rhythmicity of the cardionector) which it will accelerate or retard. This influence of the left ventricle, by means of the intramural arterial circulation, would seem to be shown by the following fact; when there is a discordance of rhythm, and the ventricular is inferior to the auricular, as in the Stokes-Adams syndrome or in flutter (see figs. 117, 167, and 175), every time that a ventricular contraction is inserted between two auricular ones, these two contractions are closer to each other than they are to their neighbors, before and behind. All happens as if the effort of the atrionector was accelerated by the action of the ventricular wave through the artery of the atrionector. This phenomenon is evidently observed only in cases in which the general rhythm, auricular and ventricular, remains constant. It may not occur if there are variations of the general rhythm. On the other hand I have shown that a case, in which the normal coördination of the auricle and ventricle was disturbed and where there was inversion of the PR sequence, the artery of the atrionector was strongly contracted. This statement is quite consistent with the hypothesis which I propose, of the influence of the arterial wave on the activity of the cardionectors. The same hypothesis explains further the variations of the interval between the auricular and ventricular contractions when the rhythm is experimentally altered. This phenomenon will be dealt with more fully later (see figs. 104, 105, and 106).

Leaving the arterial influence for a moment, we may seek to explain on the venous side, the auriculo-ventricular coördination. In this case the intervention of the right auricle is felt. It is probable that the auricle, at

the moment of contraction, retards or arrests the flow of the venous blood from the ventriculonector, which empties into the auricular cavity, and there is consequently a modification of the fluid medium bathing the ventriculonector. We may assume that to each auricular systole there is a corresponding phase of hyperemia from stasis in the ventriculonector, which determines the moment at which the ventricular contraction is produced.

The circulatory system of the cardionections, on which we hold their functioning depends, may therefore be influenced at the same time from both the arterial and the venous sides. The systole of the auricle and that of the ventricle react reciprocally upon each other by the intermediary of the parietal circulation. It is not unlikely that the auriculoventricular coördination is the result of these reciprocal actions.

The preceding considerations, however, still remain hypothetical, and an explanation of the auriculoventricular coördination, which is satisfactory in all respects, is still lacking. But I would remark that this coördination assumes a very different character, according as one studies the normal or the pathological heart. In the normal heart it appears so stable that one can imagine a fixed connection between the auricle and the ventricle in explanation of the phenomenon. In the pathological heart, on the contrary, it is the instability which strikes us, as all of this treatise will go to show. In this light the hypothesis of a fixed connection seems hardly tenable.

Thus there need be no occasion for surprise that, in the absence of a definite solution of the problem of coördination, the suggestions of the clinicians do not wholly satisfy the physiologists, and vice versa.

Action of the Nerves

Whatever theory, myogenic or neurogenic, may be adopted, all authors agree that the functioning of the heart is distinctly influenced by extrinsic nerves, the pneumogastric and the sympathetic. Weber showed that, following the application of a stimulus to the peripheral portion of the pneumogastric, a slowing of the heart's action occurred, and conversely that an excitation of the sympathetic fibers which go to the heart caused its acceleration. It would appear that the right pneumogastric and sympathetic especially control the atrionector, while those on the left side control the ventriculonector.

Uncertainty exists as to their mode of action; it is not known whether this is direct, or whether it comes about through the circulation of the cardionections as intermediary. A further problem might be to consider whether

or not the intermediary is a product of internal secretion.* We know that O. Loewi, in 1921 showed that if we excite the pneumogastric of a frog and then inject the fluid drawn from its heart into the heart of another frog, there is a slowing of the action of this second heart. Hamburger obtained the same result, as did also J. Ten Cate, later. On the other hand, Asher of Berne and Bohnenkampf were unable to confirm this action. Prof. E. Gley and Dr. P. Gley¹⁴ have shown that stimulation of the pneumogastric slows the heart, but does not act on the pancreas, provided one first divides the pneumogastric fibers which supply it. If it is through the intermediary of a product of internal secretion that the pneumogastric acts on the heart, this product which reaches the pancreas should influence its secretion. One will find in a lecture by Dr. Cardot¹⁵ and in a quite recent report of Prof. H. Frédéricq¹⁶ a detailed exposition and bibliography of works on this subject which we have only touched upon here.¹⁷

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CHAPTER IV

THE ELECTROCARDIOGRAM AND ITS VARIOUS FORMS

THE NORMAL ELECTROCARDIOGRAM

The electrocardiogram is the tracing obtained in registering the displacements of the string of the electrocardiograph, these displacements being related to the activity of the heart. The form of the tracing depends on the relation of the electrodes to the subject and to the string.

In the absence of any word to the contrary, a description of the normal electrocardiogram presupposes that the lead mentioned is Lead II, that is to say, that one of the electrodes is attached to the right arm and the other to the left leg. It presupposes further that the connections of the electrodes with the string are disposed in such fashion that the current passes through the string from above downward.

Under these conditions the normal electrocardiogram has the appearance shown in figure 39. We see the groups of electric events following each other at regular intervals. Each group comprises ascending or positive events and descending or negative ones according as they project above or below a horizontal line, called the idioelectric line. With Einthoven we designate each of these events by a letter of the alphabet. The letters P, R, T designate positive events; Q and S negative events. The letter U denotes an inconstant positive event, which sometimes follows T (fig. 40).

Lewis distinguishes in the electrocardiogram two parts which he calls the auricular complex and the ventricular complex. The former, he says, consists of a positive wave, P, followed by a horizontal line or a slightly negative wave. The ventricular complex is formed by the positive waves R and T, and two negative waves framing R, namely Q (inconstant) at the beginning of the ascending limb and S at the termination of the descending limb. The group QRS is called the group of initial events, while T forms the terminal event. Between S and T the tracing is horizontal or slightly deflected.

In reality the electrocardiogram is a composite whole which must be dissociated into an auricular and a ventricular electrogram; but this dissociation is impossible when the mechanism of the heart is normal. The terminal portion of the auricular electrogram is, in fact, confused with the initial portion of the ventricular electrogram. On the other hand, in certain

anomalies of the ventricular mechanism, this confusion ceases in numerous portions of the tracing. This is the case whenever the rhythms of the auricle and ventricle are in discordance, whether because the auricle beats faster

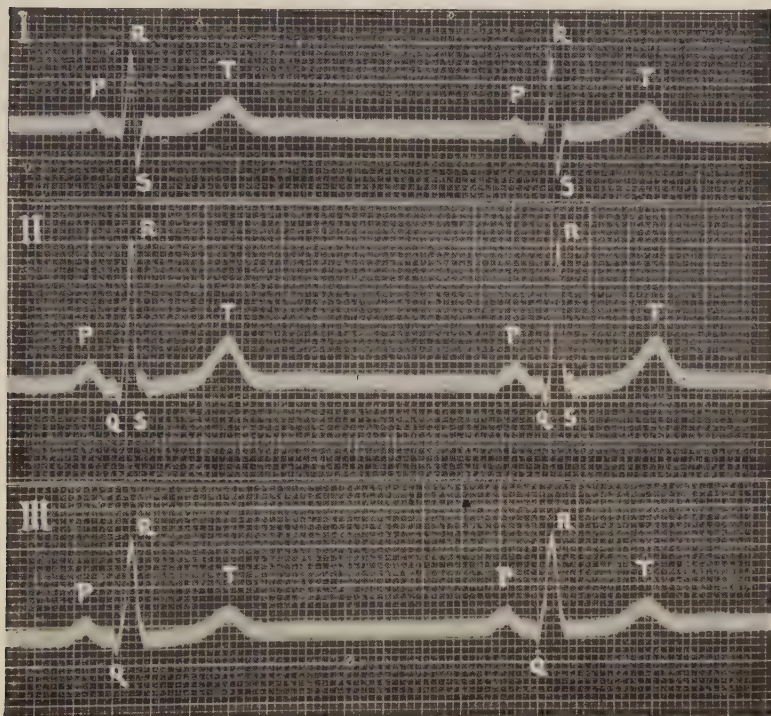


FIG. 39. The normal electrocardiogram in the three Leads I, II, III (actual size).

Note: The tracings figured in this work are reproduced without any retouching, the few exceptions being indicated in the captions. Some are of actual size, but the greater number have been reduced in order to fit the page. As they all agree in outline it is always easy to appraise the times and the electromotive forces. In every case the little square which in the original measures 1 mm., has the value of 1/10 millivolt on the ordinate, and 2/100 of a second on the abscissa.

When a tracing of unusual length has had to be divided, this has been indicated by notches, male and female, on the cut ends, so that the tracings can easily be reconstructed in the mind.

The Roman figures I, II, and III at the beginning of the tracing indicate the Leads. The Arabic figure sometimes inscribed below the Roman indicates the number of the tracing in the collection in Professor Vaquez's Electrocardiographic Laboratory at the Hôpital de la Pitié. All the tracings here reproduced are taken from that collection.

than the ventricle (discordant auricular tachycardia or flutter) or because the ventricle beats more slowly than the auricle (discordant ventricular bradycardia or Stokes-Adams syndrome).

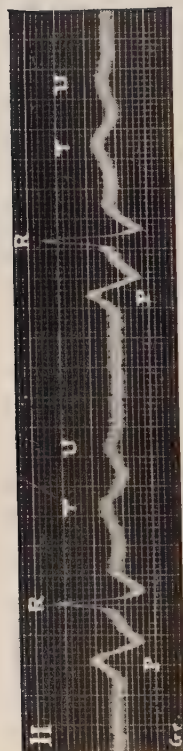


FIG. 40. The U wave in the electrocardiogram

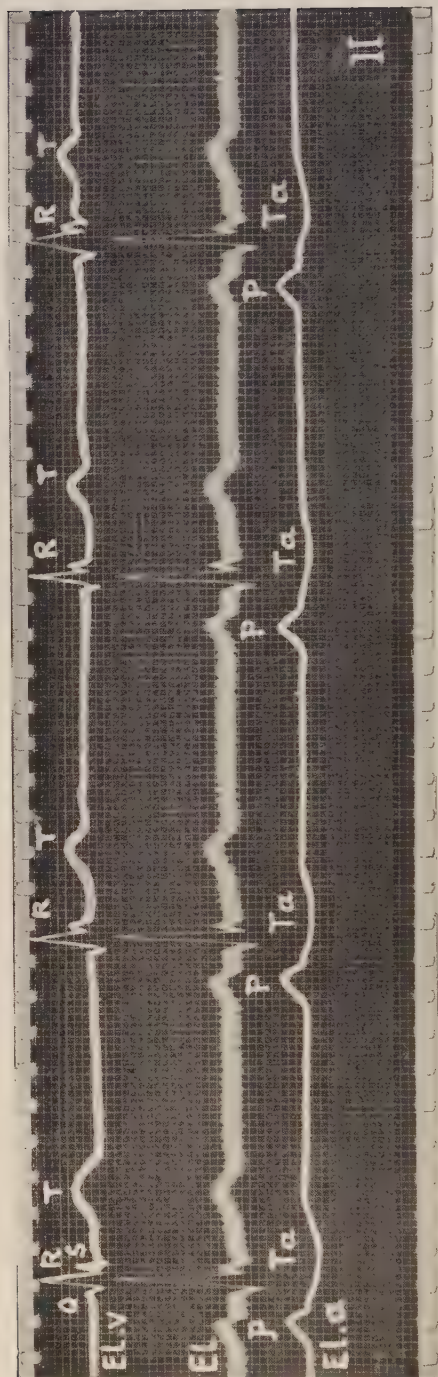


FIG. 41. The electrocardiogram EL, separated into an auricular electrocardiogram, ELa, and ventricular electrocardiogram, ELv. Reproduction in actual size of a complete film, showing on its edges the indents into which fit the teeth of the wheel which propels it. P, Ta, auricular events; QRST, ventricular events.

Experimentally, Hering¹ showed, as far back as 1908, that there is in addition to P, an event of type T associated with the functioning of the auricle. This was shown in the frog; it was seen also in a dog, whose ventricle he had caused to fibrillate under the action of curare, thus purifying the auricular pulsations. H. Frédéricq² has several times returned to this subject, and concludes from his electrocardiographic researches on the tortoise, lizard, and snake, that the wave P of the total electrocardiogram gives only a very imperfect idea of the electrophysiological phenomena which take place in the auricle. The latter, like the ventricle, has a polyphasic electrogram.

I will describe first the ventricular electrogram upon which all are in accord. It is practically the same as that which Lewis calls the ventricular complex. The ventricular electrogram consisting of the events QRST should be considered as a unit corresponding to the ventricular systole. Its length corresponds on an average, in terms of time, to 33/100 second; QRS lasts 4/100 second; the wave T takes 19/100 second (Bordet³). The time varies with the rhythm, the ventricular electrogram elongating when the rhythm is slowed and vice versa.

The auricular electrogram has considerable resemblance to the ventricular, but the auricular complex of Lewis corresponds only to its initial phase. We first distinguish the principal wave P, which is analogous to R. This event is often preceded by a negative crochet which I propose to designate by the letter Q_a from the analogy with Q which precedes R. After P the tracing takes various aspects. We follow Hering in marking with the letter T_a the wave which follows P. Drs. Howard B. Sprague and Paul White⁴ conclude from their researches on the auricular electrogram in 37 cases of Stokes-Adams syndrome, that T_a is of a direction opposite to P. Sometimes a short isoelectric line separates P from T_a . In its entire length the auricular electrogram has a duration of 34/100 to 42/100 second, or three times that of wave P. The wave T_a alone measures 22/100 to 26/100 second.

One may see by the numerous traces in the chapter on discordant auricular tachycardia that this description should be modified. Q_a is at times very marked. After P the tracing presents a strongly positive undulation, at times bristling with secondary waves; we may provisionally mark this in its totality by the letter T_a .

The auricular electrogram, quite like the ventricular comprises numerous morphological varieties. Its total duration and that of its different portions vary with the rhythm of the auricle. A more precise description would be premature. I wish here only to draw attention to the existence of an

auricular electrogram from which, in the classical descriptions, the second half is generally amputated.

The Electrocardiogram in Leads I and III

The preceding description was that of a tracing of Lead II. When we introduce another lead the electrocardiogram changes its form. It will be remembered that Lead I is that in which the electrodes are placed on the right and left arms; in Lead III they are in connection with the left arm and left leg.

If we take account solely of the initial undulation of the ventricular complex and compare the forms it assumes in passing from Lead I to Lead II and Lead III, the existence of three types may be noted:

First, or ascending type; the negativity of the initial wave diminishes or its positivity increases (fig. 46).

Second, or descending type; the positivity of the initial wave diminishes or its negativity increases (figs. 42, 43 and 44).

Third, or mixed type; this type presents two varieties, in one of which (fig. 45) the undulation is ascending from Lead I to Lead II, and descending from Lead II to Lead III; in the other it is descending from Lead I to Lead II and ascending from Lead II to Lead III.

It may happen that we may have only one tracing in a single lead, either I, II, or III. It is generally regarded (which is often the fact, but not always) that Lead II is characteristic of the type. It is also admitted that the initial wave has the same direction in Leads II and III, but an opposite direction in Lead I from what it has in Lead II.

When the initial wave is positive we say that there is a right preponderance; when it is negative, that there is a left preponderance. This would perhaps imply that the preponderance of one or the other ventricle exerts an influence on the electrocardiogram. This is far from being invariably true.

Einthoven (fig. 46) has stated that if one measures the heights of R in the three leads these will correspond to the equation $R_{II} = R_I + R_{III}$. This author has shown further that one can obtain from the deviations of the string produced at the same instant in the three leads precise indications as to the amount of the electromotive force manifested at this instant and of the direction of that force. But evidently this does not give the true measure of that force, and even if it did, we should have in reality only the algebraic sum of all of the individual forces. Tables have been constructed which permit us to deduce from the values of Lead I, Lead II, and Lead III the amount of the manifested electromotive force (EM) and the angle

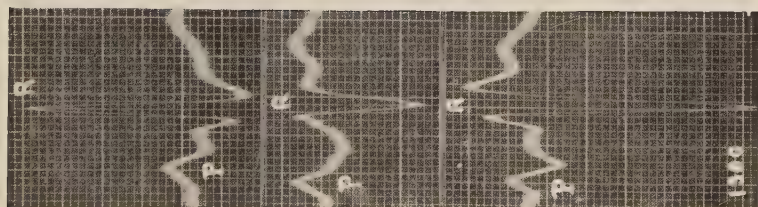


FIG. 42

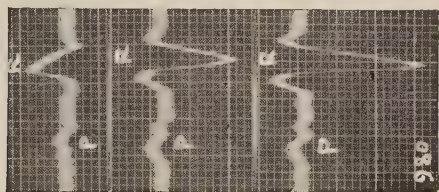


FIG. 43

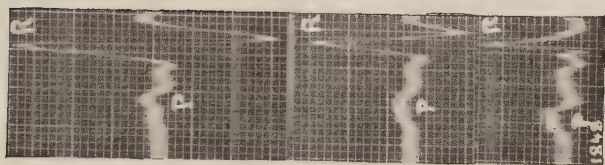


FIG. 44

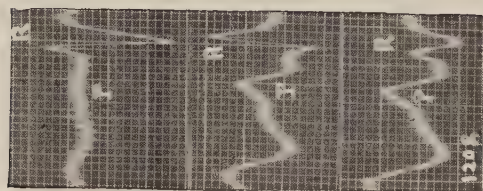


FIG. 45

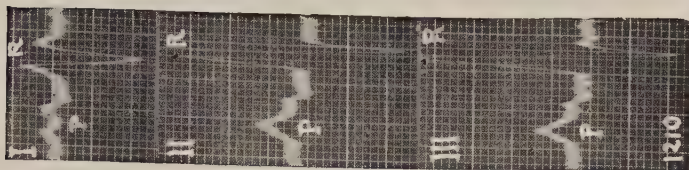


FIG. 46

FIGS. 42 TO 46. Some varieties of the ventricular electrocardiogram in the three leads. Figures 42 to 44, descending type; figure 45, mixed type (ascending-descending); figure 46, ascending type.

which the direction of this force makes with the horizontal or angle α . We will not pursue these considerations further, and can only refer the reader to the works of Einthoven, Fahr, and Waart,⁵ Pardee,⁶ Carter, Richter, and Greene,⁷ Dieuaide,⁸ and Arthur Weber.⁹

Synchronism of the Events in Leads I, II, and III

When the tracings in the three leads are registered simultaneously, it is noted that there is no absolute concordance between the events. The result is that a certain doubt often persists as to the identification of the events of the different leads. In a general way if one compares the tracings in Lead II and Lead III with a tracing taken in Lead I, it will be seen first that P II and P III are slightly anterior to P I. Second, that the groups P II—R II and P III—R III are slightly shortened in comparison with group P I—R I. The differences which exist between the stages of these homologous events in the three leads are in reality very slight. Nevertheless they tend to make difficult the homologation of the events. Moreover, if, as we shall see, the electrocardiac curve is only an algebraic curve, it is sometimes illusory to seek to find in different leads the same electric event. Thus it often happens that the notation is perhaps quite artificial. When in one lead we find an initial ascending peak, and in another lead an initial descending peak, we mark the first with the letter R, the second with Q or S. This is in conformity with the convention adopted since the work of Einthoven; but this does not always prove that the Q of one lead corresponds to the Q of the other lead. Besides it is entirely anomalous to designate with the same letter P the initial event of the auricular electrogram, even when this event changes direction, since one does not do the same in the case of the initial event of the ventricular complex R.

For researches of precision and especially for those which have for their end the elucidation of the causes which alter the form of the electrocardiogram, all of this nomenclature would have to be revised. Our work, which will only graze the subject of the morphology of electrograms, has, on the contrary, to take into consideration only the principle events of the curve, P, R, and T, without specifying minutely what happens when one changes the lead, for the error cannot amount, at the most, to more than one or two hundredths of a second—a negligible error in studies of the cardiac mechanism.

Change of the Connections of the Electrocardiograph

If one inverts two by two the wires running from the electrodes, in relation to the poles of the apparatus, the electric events, for the same lead

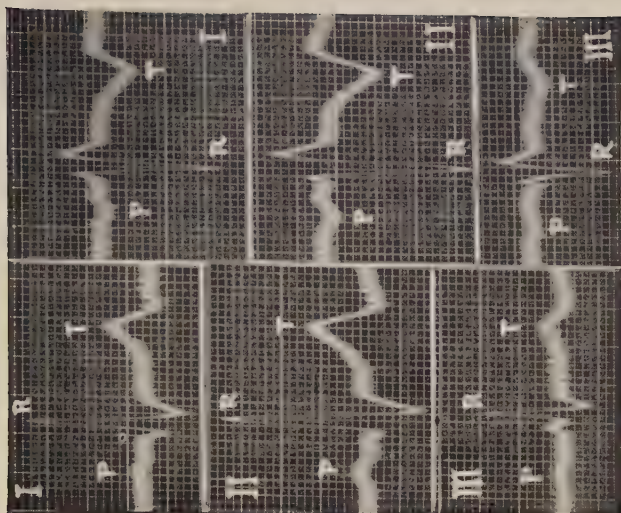


FIG. 48

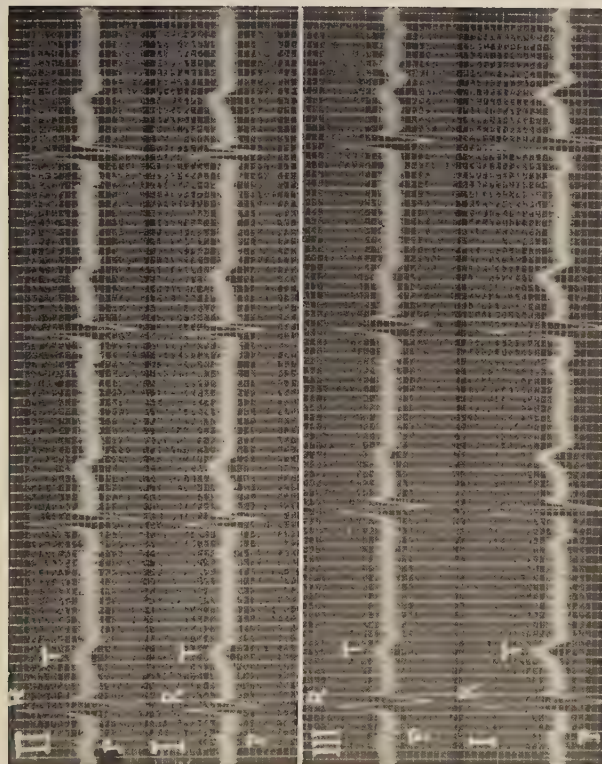


FIG. 47

Fig. 47. Synchronism of the events. The four tracings were taken, two by two, by means of an electrocardiograph with two strings which I owe to the courtesy of the Boullite firm. Exceptionally the chronograph here marks 1/10 and 1/20 second.

Fig. 48. Normal and reversed electrocardiograms in the three leads. The three leads to the left of the reader are taken with the usual connections; the three leads to the right are taken by reversing the electrodes two by two, for example, connecting the right arm with the terminal BG of the control board, the left arm with the terminal BD.

change their direction. The explanation of this is that the inversion causes the current passing through the string to change its direction, and the R from positive becomes negative, the S from negative becomes positive, and so on for all the events of the curve. The curve thus obtained is called the "mirror curve." We find this same mirror curve if it is the heart of the subject which is inverted in relation to the two ends of the electrocardiograph. This is the case where there is a total transposition of viscera, the heart and aortic arch being on the right side. But the mirror tracing exists only in Lead I. For the other leads, II and III, the tracing differs slightly, but there is no change of direction. In dextrocardia without total transposition of the viscera, there is no mirror tracing, whether in Leads I, II, or III, for though the axis of the heart is more or less inclined on the vertical, it does not, as in total transposition, take a symmetrical position in relation to the vertical.

EXPLANATION OF THE ELECTROCARDIOGRAM

There is no satisfactory explanation of the electrocardiogram, but nevertheless it will be useful to review briefly all of the hypotheses proposed, because of the suggestions which each awakens. In general, only the ventricular electrogram QRST is considered, and it is with this that the present chapter will deal.

One fact is well established, namely, the electrical phenomena take place when the heart contracts, and in the interval of the contractions there is no longer any electrical manifestation. To what do these electrical phenomena correspond?

The first question to solve is whether all of the electric phenomena are connected with generating causes of the same nature. If it is only a matter of quantitative variations, the electrogram is a simple curve. If, on the other hand, it is a matter of an ensemble of electrical phenomena, some of which, for example, correspond to a phenomenon of excitation prior to the contraction, others to a mechanical phenomenon of contraction, and others again to a phenomenon of change of form when the muscle contracts and then relaxes, the electrogram is a composite curve.

THE ELECTROGRAM IS A SIMPLE CURVE

Einthoven and a number of authors before him have stated that the ventricular complex is a simple curve, all of the events of the complex representing quantitative variations of one and the same phenomenon. What is this phenomenon?

The Electrogram Indicates the Advancing Wave of Contraction in the Myocardium

It is classical to recall in this connection phenomena observed when the gastrocnemius of the frog is caused to contract. We will not detail them but will merely recall the generally admitted facts that every movement of living matter, muscle in particular, gives rise to a difference in electric potential between the active part and the part which remains inactive. This difference of potential necessarily determines a current, a current of action (Hermann).

If then we consider the ventricular mass as a muscle, we reason thus: as all the mass does not contract at the same time, the part which does contract becomes negative, the part still in repose remains positive, and an electric current is produced. And since the different parts of the ventricles come into play successively, there is a variation in the direction and intensity of the current produced. The electrogram would then reflect the existence of these changes in the current.

But we know little about the functioning of the ventricles, and authors are not in accord as to the explanation of what transpires in their interior. Some tell us that there is first a contraction of the base, then one of the apex; others say that there is first a contraction of the columnae carnae, then of the apex, then of the base. This would appear to be more in relation to the result, if not the purpose, of the ventricular systole, to wit, the expulsion of the blood into the great vessels. Moreover, the disposition of the two ventricles with their respective axes crossing almost perpendicularly, detracts much from the value of the expressions base and apex. Finally the character, in a way explosive, of the ventricular systole does not tally well with the relatively long duration and the spacing of all the electric events of the electrogram.

This theory, which relates the electric phenomena to the contraction of the myocardium is with difficulty reconciled with the fact that it has been found possible to obtain an electrogram from an arrested heart. We may recall on this subject, following Weber, the researches of Trendelenburg in 1903, that of Noyen in 1908, of Straub in 1910, of Samoiloff in 1914, of Mines in 1915, of Klewitz in 1917 (heart arrested by muscarine poisoning), of Bergsanius and Bijtal in 1914 (deprivation of calcium), of Hegler in 1912 (electrogram obtained in a human subject half an hour after death). More recently Henrijean¹⁰ obtained an electrogram from an arrested heart, the arrest being proven by means of the muscular telephone of d'Arsonval.

The electric phenomena then do not depend on the muscular contraction

since they have been observed in a heart which is not contracting.* If they are observed synchronously with muscular contraction, although these do not depend necessarily one upon the other, it is because both depend on the same cause, whence their synchronism. This common cause is the advancing wave of excitation.

The Electrogram Indicates the Advancing Wave of Excitation

We know nothing of the nature of this process of excitation. For some it is a stimulation of nervous origin arising in the autonomous ganglia of the heart; while others regard the stimulus as originating in the myocardium itself. In one hypothesis as in the other, whether the excitation comes from above or whether it is produced in the auricle itself, a wave of excitation from the auricle will follow the course—auricular myocardium, atrio-ventricular bundle, ventricular myocardium.

The electric phenomena set free by the passage of this wave in its peregrinations cause the production of currents of which the electrogram expresses the variations.

We may admit with Lewis, who particularly maintains this hypothesis, that the wave of excitation is propagated with a velocity which varies in different sections of the area traversed. This is one meter a second for the auricular myocardium, two meters in the atrioventricular bundle, and forty centimeters in the ventricular myocardium. Lewis also believes that the excitation, and consequently the electric manifestation, precedes the contraction by a measurable interval.

These statements of Lewis have been criticized as follows: (1) There would be no precedence of the electrical phenomena in relation to those of mechanical nature; or if there were it would be insignificant, being a matter not of hundredths of a second, but at the most of thousandths. If the precedence were apparent, it would be due, according to Einthoven and de Jongh,¹² to an insufficient sensitiveness of the apparatus for the registration of mechanical phenomena. With sufficiently sensitive apparatus these authors have been able to show a synchronism of electrical and mechanical phenomena. But Weber, in his turn, opposing the assertion of Einthoven

* Some have based their argument on the observations of Spadolini and Giorgi, which I quote from Padilla.¹¹ These authors obtained galvanometric deviations in the heart of a chick embryo, at the 50th hour, that is to say, prior to any differentiation of muscular fibrils; but this does not mean before any contraction occurred, for it is not a question of morphological differentiation, but of functional capacity, and there is no doubt that the vascular tube of the embryo contracts. This observation neither confirms nor invalidates the preceding statements.

and de Jongh, says that the electrical precedence, in which he believes, escaped them because the galvanometer string lacked sensitiveness.

(2) The figures given by Lewis for the rapidity of the wave of excitation are erroneous, according to Athanasiu. A speed of one meter a second means for one centimeter of auricular tissue $1/100$ second. In the auricle of a dog the septum and apex are 5 cm. apart; so that if the electrograms of these points were registered there would be a difference of $5/100$ second; but the synchronism is perfect. There is also synchronism in the electrograms of the apex and of the base of a ventricle, and likewise synchronism in the electrograms of the two ventricles. One cannot therefore accept the opinion of Lewis without reserve, and I would add that it is difficult to comprehend just what this wave of excitation corresponds to and what is its cause.

The Electrogram Interprets the Phenomena Closely United with the Physiochemical Modifications which Cause the Myocardial Contraction

The mechanical phenomena of the contraction result probably from preceding physical phenomena. It is these that are manifested electrically. The electrocardiogram of the arrested heart would thus be explained, since the primary molecular modification would take place with its electrical manifestations and only the mechanical phenomena would be lacking. In this connection we recall that Henriques and Lindhard in 1920 obtained a powerful contraction of the gastrocnemius of the frog, without the least current of action being registered, when the muscle was directly excited. On the other hand they registered a current of action by indirectly exciting the muscle through the intermediary of its nerve. It would seem as if the stimulus, when it reached the muscle through the intermediary of the nerve had to go through a stage of transformation in order to become efficient, the passage to this stage entailing an electric manifestation. If the stimulus reaches the muscle without passing through the nerve, and therefore without a stage of transformation, the contraction takes place but there is no electric manifestation.

Deglaude¹³ has recently proposed the following explanation: As an effect of the stimulation there are molecular modifications which give rise to an electromotive force. One part of this force is transformed into the work of contraction, while the other, not transformed into energy, gives origin to a current. The phase of the charging is very rapid and corresponds to the initial events, while the phase of discharge would be very slow, corresponding to the event T. Deglaude recalls in support of this hypothesis the observation made by Henrijean. This author showed that in the electro-

cardiogram of an arrested heart the height of T is equal to that of R. According to Deglaude the electromotive force is here entirely transformed into current, for no contraction takes place. The current of discharge is then equal to the current of charge.

All of the preceding hypotheses admit the intimate connection between electric phenomena and phenomena of contraction, differing only as to the manner of connection. In all the hypotheses the attempt has been made to be more precise in relating the different events of the ventricular complex to the different times of contraction. Arthur Weber sums up these relations as follows: "The excitation is diffused in the specific system from the pillars toward the apex during the event Q, from the pillars toward the base during the ascending portion of R. The descending portion from R to S is due to the propagation of the stimulus through the myocardium proper and in particular that of the apex. While the curve mounts up from S there is a growing extension of the excitation in the basal portions of the myocardium, the apical preponderance diminishing accordingly. In the part of the tracing which follows, all of the heart has the same negativity up to the point, where, with the entrance of T, there is a recession of the excitation, first at the apex, and later at the base."

This explanation reproduces in substance that given by Einthoven in 1912 and differs but slightly from that of Nicolai. This last author, however, considers that the phase of propagation of the excitation from the base to the apex through the intermediary of the bundle of His and the papillary muscles corresponds to PR. RST corresponds to the contraction of the middle, circular, layer of myocardial fibers; T to the contraction of the spiral fibers.

Gotch¹⁴ says there would be at first a wave going from the base to the apex, whence the events QRS; then a wave going from the apex to the base, T, this event being more particularly the evidence of the energetic terminal contraction of the bulb of the aorta.

Kraus and Nicolai¹⁵ think that each of the events of the electrogram corresponds to the activity of one particular layer of the ventricle; R to the contraction of the papillary muscles; the interval between R and T to the contraction of the middle layer of the myocardium (the "Treibwerk" of Krehl), and T to that of the spiral fibers. It is in following this theory that Kraus and Nicolai have given to the events of the electrogram a different explanation from that of Einthoven.

Without insisting further on the uncertainty concerning the functions of the different portions of the ventricle we may remark with H. Frédéricq¹⁶ that it appears very illusory to expect to find in all the events of the electro-

gram a manifestation of the activity of the different portions of the ventricle. The electrogram of mammals resembles that of the cold-blooded animals, and yet the structure of the heart is very different.

THE ELECTROGRAM IS AN ALGEBRAIC CURVE

We may regard the events of the electrogram as not individually representative of the diffusion of a wave (of physical transformation, of contraction or of excitation) passing to different portions of the ventricular myocardium. The electrogram is a curve representing the algebraic summation of variations of potential produced at a given instant in all parts of the heart.

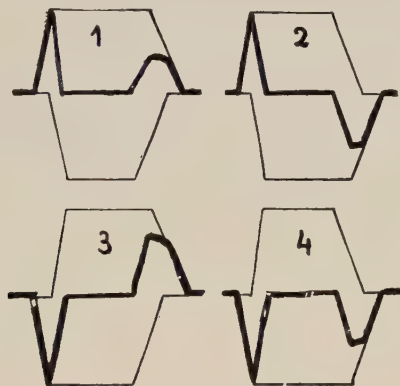


FIG. 49. de Boer's schema. Two curves of the same form but in contrary directions (fine lines), in combining differently give as the algebraic sum the curves, 1, 2, 3, 4 (coarse lines) analogous to the different types of the ventricular electrocardiogram.

In 1912 Einthoven,¹⁷ in propounding his theory of the electrocardiogram, wrote: "The difference of potential shown at a given moment by the galvanometer is the final result of the differences of potential which exist at the same instant in different portions of the heart. One may divide the heart into any two portions and the electrocardiogram will still be the result of the variations of potential existing in these portions. The curve should evidently represent at each instant the algebraic sum of all the variations of potential which are developed in the heart at the same moment."

Thus considered, the electrogram is a polygram and it has especially been interpreted as a bigram, following the expression of Lewis; a bigram behaving like an electrogram of the base and of the apex, following the conception of Einthoven as restated by Bohnenkampf; or an electrogram of the right ventricular mass and left ventricular mass following the hypothesis of

Selenin;¹⁸ or finally an electrogram of the right and left branches of the bundle of His, according to the theory of Lewis. In these related conceptions one of the constituent parts of the electrogram may be predominant and the total electrogram undergo a corresponding change in form. There would be right or left preponderance, basal or apical preponderance, or an electrogram of the middle layer of myocardiac fibers ("Treibwerk" or Krehl), or of the longitudinal fibers, according to the hypothesis of Eppinger and Rothberger.¹⁹

We reproduce here the schema of de Boer,²⁰ analogous to that of Bohnenkampf (fig. 49). It is interesting and suggestive because it shows what a complex form the algebraic curve may take when it results from the interference of two phenomena, of which each, when isolated, is interpreted by a simple curve, increasing and then decreasing. Now the simplicity of each of these curves is better comprehended if we regard each curve as indicating the electric variations in relation to the phenomena of contraction which increase and decrease them regularly, as shown in Marey's curve of the intraventricular pressure.

Bohnenkamp says that the electrogram is a curve of algebraic addition of two opposed monophasic currents acting on the string of the galvanometer, one current coming from the base and the other from the apex.

deBoer holds that the electrogram results from the interference of the negativities of the base and apex dissociated from one another by the time factor. This dissociation varies with the rapidity of conduction, whence the variations in the interference.

THE ELECTROGRAM IS A COMPOSITE CURVE

In the preceding theories the electrogram with all its events is considered as a simple curve, algebraic or not, which interprets quantitative variations of electrical phenomena having a common origin. In the theories which we are about to enumerate the electrogram is considered as a composite curve, the events of which depend on electrical phenomena having different origins. Generally, in the ventricular complex, the initial events, QRST, are opposed to the terminal event, T.

Hoffmann²¹ and Seeman²² claim that the initial events, QRS, express the conduction of the stimulus and corresponding to the current of action of the striated muscles. "The terminal event, T, may be regarded as at least a manifestation which accompanies the process of contraction." QRS on the one hand, T on the other, would not then be the two phases of a single current of diphasic action. Likewise Eyster and Meek²³ see in T a wave connected with the contraction. According to Eiger,²⁴ β (the isoelectric

line between S and T) and T do not respond to a current of action but to biochemical processes. H. Straub holds a similar theory.

Meyer²⁵ thinks that the events QRS and the ascending limb of T interpret currents of action; the apex of T corresponds to the end of the systole. Its descending branch and U, which is often seen following T, would then correspond to currents due to the change of form of the myocardium in relaxation.

H. Frédéricq²⁶ finally, holds that the events QRS represent the rapid contraction of the muscular fibrils, while T corresponds to slower and more persistent contraction of the sarcoplasmic mass. In addition to the preceding theories we may note further the hypothesis of Ussow.²⁷ In accord with the majority of authors, he considers QRS as the expression of the contraction of the ventricles; but he gives an entirely different explanation of T which he says is in relation with the change of form and of position of the heart considered in its totality and its respective parts. Following the contraction the current which passes through the body will be modified there. But this ingenious hypothesis, thus presented, clashes with the fact that T exists in the electrogram of the isolated heart. Perhaps it will not be necessary to reject this hypothesis entirely. We may consider that the electric phenomena are localized in the specific myocardium. These phenomena externalize themselves only in passing through not only the latter, but also the enveloping contractile myocardium. We may conceive then that their manifestation may be modified if the form and thickness of the myocardium are modified; and in effect they are modified. We shall return repeatedly to this hypothesis.

MORPHOLOGICAL VARIETIES OF THE ELECTROCARDIOGRAM

The Auricular Electrocardiogram

We need only study the tracings given in this work to recognize the various forms which the auricular electrogram may present. Let us recall in this connection that we must not regard this curve as consisting solely of P, for this is only the initial event. We shall see some of the varieties of this electrogram in the chapter on auricular tachysystole. P seems to be usually monophasic and positive, but it is frequently diphasic, with the first phase either positive or negative. Also cases are met with in which P appears as monophasic and negative, P being inverted. This negative P has been interpreted as indicating a systole of the auricle, due to an abnormal stimulus passing up from the node of Tawara to the auricle. This is purely an hypothesis. Cases like that figured in the following chapters (figs. 104,

105, 106, 107), in which P, at first positive, becomes progressively negative, then again positive, do not fall at all in this category. When P appears in the midst of the ventricular complex, between R and T, it changes its direction, but this inversion occurs in only a very limited portion of the interval RT. The same peculiarity is frequently (but not constantly) encountered in cases of auriculo-ventricular anisorhythmia from discordant ventricular bradyrhythmia. We believe that the change of form of the auricular electrogram results from its coincidence with a different phase of the change of form of the ventricular myocardium; as if, in other words, one "saw" it through a differently shaped myocardium.

The auricular electrogram may undergo progressive modifications which change its form considerably. It is a question of modifications analogous to those offered us by the ventricle (see fig. 152).

The bifidity of P is without significance. This anomaly is seen in the antepenult complex of the tracing (fig. 152), as also other changes of form which are insensibly related one to another. This bifidity has at times been considered as a proof of the discordance of the systoles of the two auricles, but it is generally admitted today that there is an absolute synchronism between these two cavities.*

The Ventricular Electrogram

It has been known for a long time that the ventricular electrogram may undergo a notable change of form in relation with the respiratory movements. Professor Vaquez and Dr. Bordet²⁸ have published typical curves on this subject, of which the following (fig. 50) is an example. In the phase where the ventricular electrogram is most developed the subject made, on request, forced respiratory movements. The preceding phase corresponds to the period when he was breathing quietly. The parallel variation of the pressure curves of the brachial and tibial will be noticed (fig. 50). In certain individuals the respiratory movements, even without being forced, influence the complexes considerably, especially the ventricular (fig. 51). One may note also the variations of shape of the ventricular complex in relation to the position of the subject, whether in the dorsal or lateral decubitus, and with the modified shape of the abdomen in the pregnant woman. Every change of relation between the heart and the thoracic envelope influences the morphology of the ventricular complex, although such variations are inconsiderable.

* H. Frédéricq sustains the contrary opinion and shows tracings in its support, but he does not take into account the experiments of Athanasiu.



FIG. 50. Influence of forced respiration on the ventricular electrocardiogram. Of the two blood-pressure curves registered at the same time as the electrocardiogram, the upper one, from the tibial artery, is the only one visible in the tracing, the lower one, from the brachial artery, was very imperfectly reproduced.

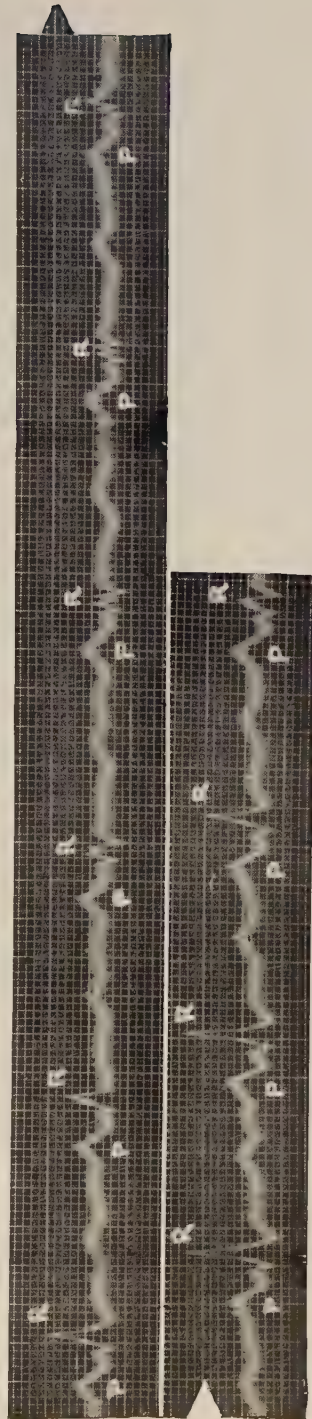


FIG. 51. Influence of quiet respiration on the ventricular electrocardiogram. Note the variations of the initial wave R in the ten registered beats (Lead II).

It has also been observed that the ventricular complex presents a difference of type according to whether there is a preponderance of the right or left cavities, the initial events being of the ascending type in the three leads when there is a preponderance of the right cavities and of the descending type when the preponderance is of the left cavities. But this is not an absolute rule and there are numerous cases in which the left ventricle, plainly hypertrophic on the screen, does not give the type qualified as of left, but, on the contrary, that of right preponderance.

Prof. Vaquez and Dr. Bordet, in their report previously cited, distinctly assert that "practically it is not possible to depend exclusively on the curves of the electrocardiograph in the diagnosis of hypertrophy with predominance of the right or left ventricle."

I will add that, after an interval of several days, one may observe a complete modification in the type of the ventricular complex, even in the same lead, of such sort that the positive of the outset of the complex becomes negative, to return again to the positive. It is possible that this variation of complexes may be dependent upon a variation in the respective volumes of the cardiac cavities and especially of the auricles.

Change in Form of the Complexes in Pulsus Alternans

We know what is the phenomenon of alternating pulse—the radial pulse is alternately strong and weak. If we take at the same time a tracing of the arterial pressure and an electrographic tracing we find that in a certain number of cases there is alternation also of the electric complexes.

Electric alternation is seen habitually with rapid rhythms. It is clear in the tracings reproduced in the chapter devoted to auricular tachyrhythmia (figs. 114, 127), and I give here a typical tracing (fig. 52). The electrical alternation is clean cut, while alternation of the arterial curve, although present, is less marked. We see that the feeble pulsation corresponds to the ample complex, but the converse may sometimes be observed. Electrical alternation is especially manifest in the ventricular complexes and shows in all the events of the same, in R, S, and T. R is the event in which the modification of the height is most striking, but we may see in the tracing that alternation is very evident also in T (fig. 54).

The tracing in figure 53 furnishes another example of electrical alternation, of interest because of the two proisystoles which, having broken the ventricular rhythm, break also the alternation. In this subject with the rapid pulse continuing at the same rate the alternation is sometimes present and sometimes absent.

Electrical alternation may occur, however, with a rhythm practically



FIG. 52. Electrical alternation and alternation of the blood-pressure curve in the brachial artery. The auriculo-ventricular beat is 200

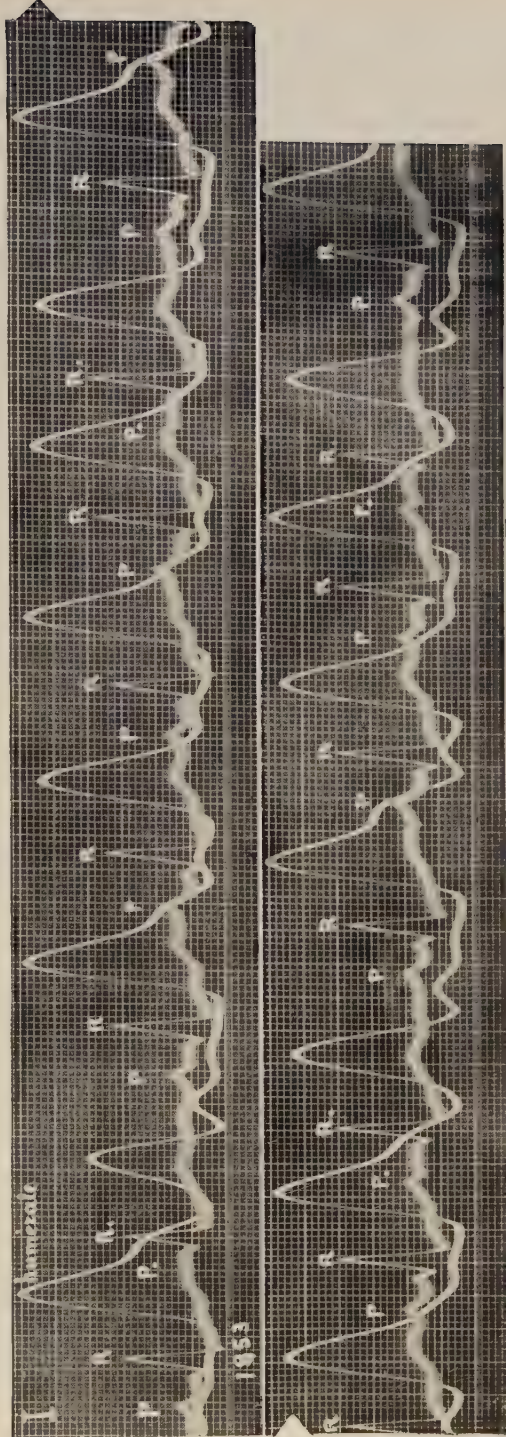


FIG. 53. Electrical alternation broken by the auriculo-ventricular protosystole. Rhythm, 120. The electrical alternation, which is distinct at R_3 , R_4 , R_6 , R_8 , is broken at R_7 ; the alternation reestablished at R_{12} , R_{13} , R_{14} , is again broken at R_{15} . R_7 and R_{15} indicate ventricular premature systoles or protosystoles. The alternation of the brachial curve is also broken, but only once; the arterial wave corresponding to a premature systole is always a low wave and if the curve of alternation regularly comports with a low wave at the time the premature systole occurs, the alternation is maintained; this is the case with the wave corresponding to R_{15} . But if the tracing should at that time show a high wave, the alternation will be broken; this is the case with the wave corresponding to R_7 .

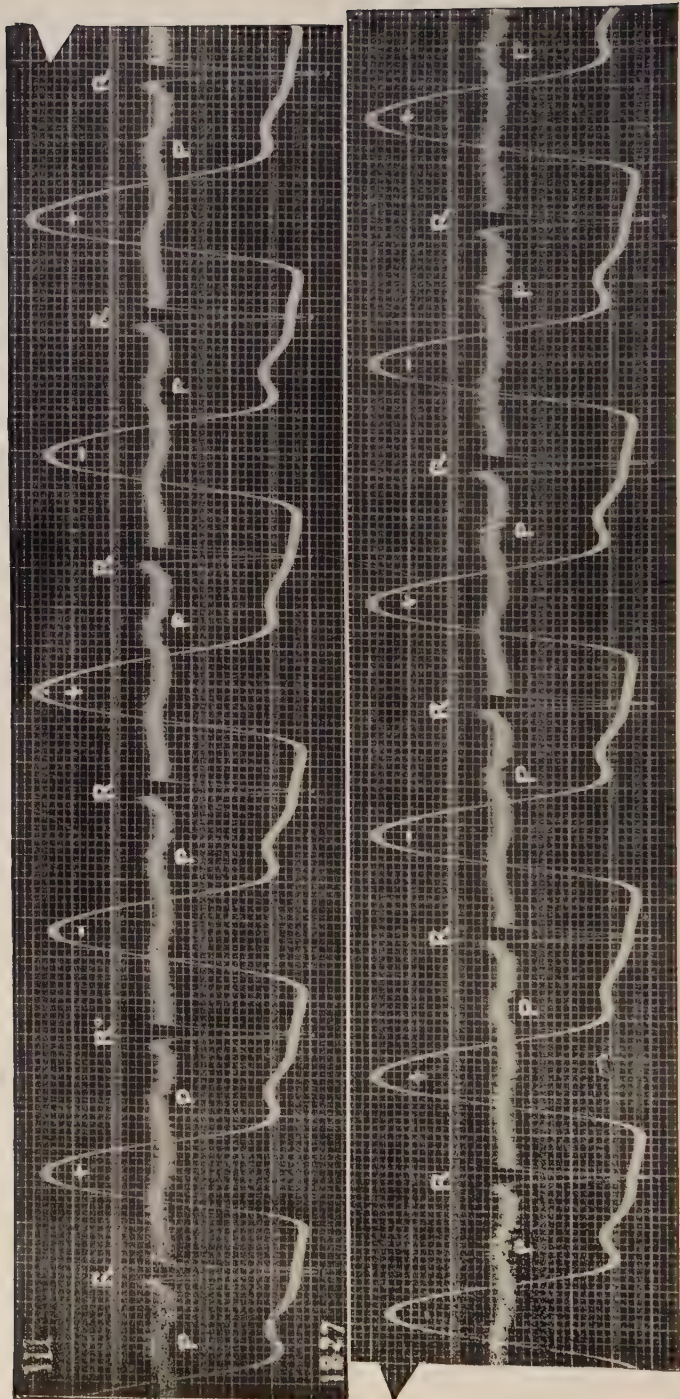


FIG. 54. Electrical alternation and arterial pulse alternation with a slightly accelerated rate, being only 95. The electrical alternation is evident at the level of the first four R waves. It is characterized especially by the height of the first ascending event, alternately higher and sharper and lower and more rounded. The length of the descending arrow which follows does not show a very marked variation from the pulse rate. The alternation of the arterial pulse is much more distinct, but it becomes considerably less marked in the last five waves which are practically of the same height. Note that at the beginning of the curve the alternations are not opposed to each other, as in figures 52 and 53 where, corresponding to the ample ventricular electrogram there is a weak pulsation, and vice versa.

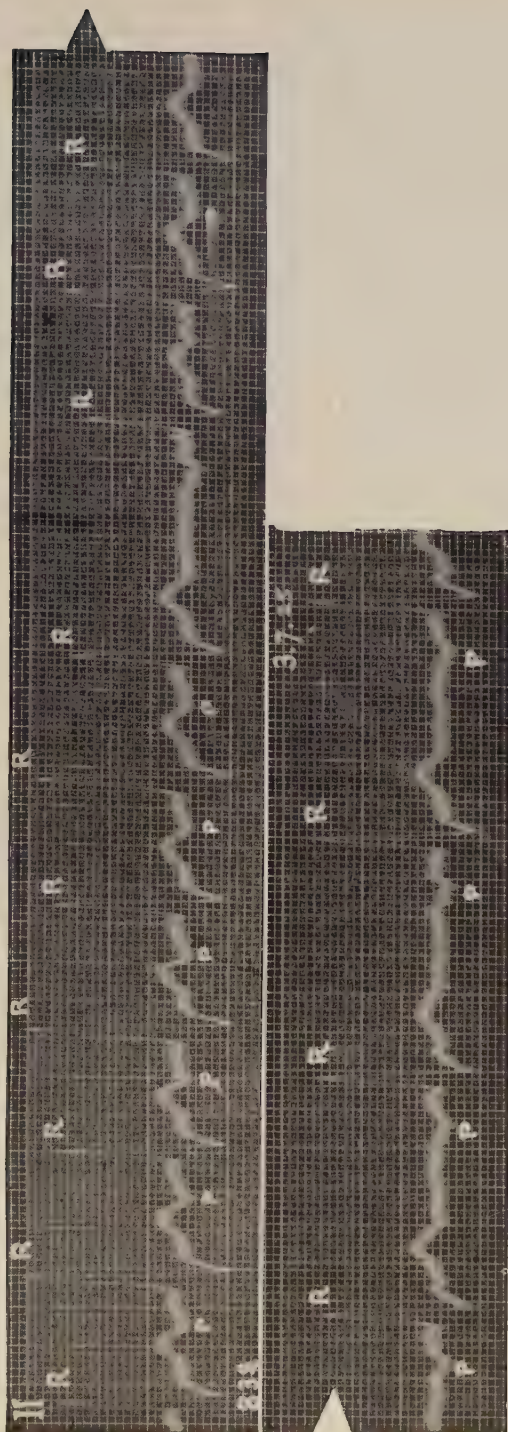


FIG. 55. End of an attack of auriculo-ventricular tachycardia, an abortive new attack, and return to the normal rhythm. The alternation, very distinct during the attack, reappears at the abortive attack, and disappears on the return to normal rhythm.

normal. The tracing in figure 54 shows this alternation with pulse rate of 95 and on this tracing the attenuation of the alternation may be noted. We see on the tracing in figure 55 that the ventricular complexes alternate during the crises of coupled auricular and ventricular tachyrrhythmia, alternate again at the moment of the abortive outline of a second crisis but cease to alternate when the normal rhythm returns. On the tracing in figure 56, the electrical alternation, accompanied at first by alternation of the pulse, continues alone during the greater length of the tracing. A somewhat similar variation is shown in figure 143.

Several explanations have been given of *pulsus alternans*. The fact that alternation of pulse and electrical alternation do not always coexist and that the two behave differently when both are present—one strong pulsation corresponding sometimes to a low and sometimes to a high complex—shows that it is necessary to explain these two phenomena separately and to distinguish alternation of the pulse from electrical alternation. The theories which have been proposed do not separate these two subjects clearly. What they seek to explain is only alternation of the pulse. We may instance in proof of this statement the theories of *hemisystolia* and of *periodical mitral insufficiency*, the exposition and criticism of which will be found in the works of Professor Vaquez.²⁹

Gaskell³⁰ and Hering³¹ hold that in the case of alternating pulse the heart does not contract in its entirety but only in part. The volume of the contracting portion varies alternately at each pulsation, so that a strong pulse corresponds to the maximal volume; a feeble pulse to the minimal. The absence of contraction of the inert parts of the myocardium is explained on the supposition that they require more time to escape from their phase of non-excitability. This theory has been criticised by H. Frédéricq.

Wenckebach³² interprets the strong pulse as due to a strong cardiac systole, the weak pulse to a weak systole, this difference in the systoles being determined by a disturbance of rhythm. After a *proiosystole* a prolonged diastolic pause brings about a more complete filling of the ventricle and as the result the first systole is a strong one. But the pause which follows is brief, there is a less complete filling and the second systole is weak. Wenckebach assumes that some of the blood remains in the ventricle and that to this blood is added that which flows in during the pause, so that the ventricle, now more distended, will react by a third systole which is strong. We confess that we do not comprehend why any blood should be left behind in the ventricle, nor, on the other hand, why, after a third strong systole, the fourth one should be weak. We fail to see the reason for this, since now there is no variation in the diastolic pause. In all these theories we visualize

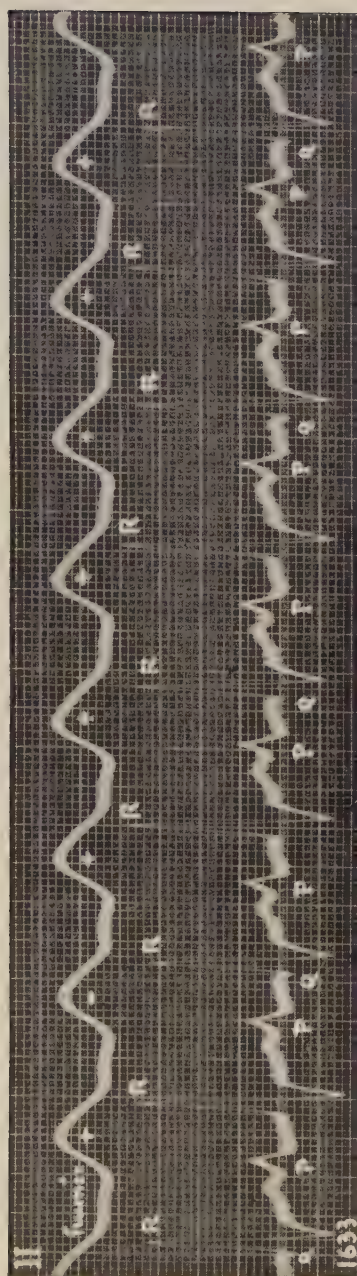


FIG. 56. Electrical alternation accompanied, at the beginning of the tracing only, with alternation of the pulse

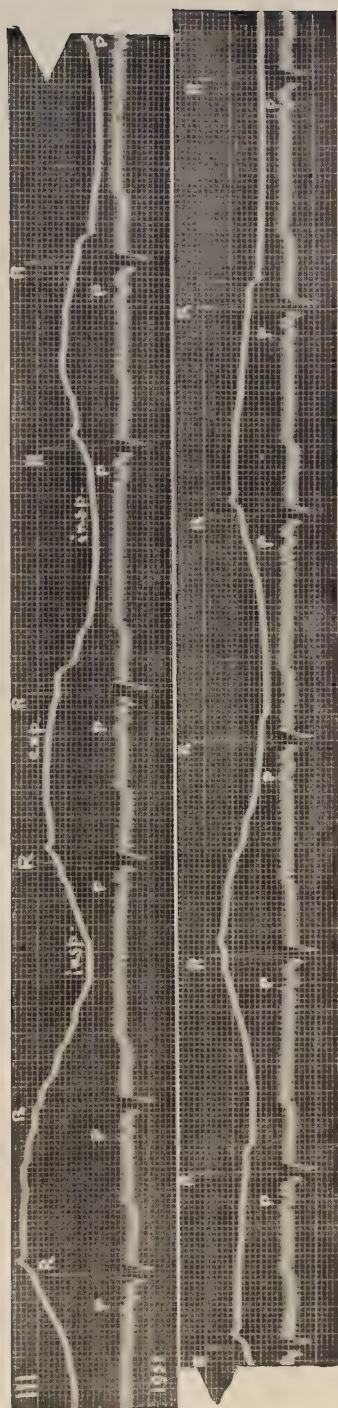


FIG. 57. Electrical alternation in close relation with the succession of the respiratory phases

as a factor of the pulse only one element, the myocardium. We incriminate now its degree of conductivity, now its contractile force, and now the duration of its refractory phase. But Straub's theory³³ introduces a second element, the pressure in the interior of the vessels. Weber, in summing up this theory, states that there is alternation when the frequency of the systoles is so great and the fall of intravascular pressure so slow, that a new systole is produced before the pressure has fully descended. We agree with Weber that none of these theories is satisfactory.

We understand alternation otherwise. It appears when the circulatory disturbance which determines all perturbation of rhythm, is not immediately compensated. When the ventricular series is interrupted by a premature, isolated systole, this systole launches a wave of blood of slender volume, the diastolic filling of the ventricle having been curtailed.³⁴ The arterial curve is depressed, then if the following systole is not premature it is raised to its original level. If the premature systole inaugurates a crisis of tachyarrhythmia, the arterial depression persists but the tension, for a moment lowered, is soon reestablished. However, the time of diastolic filling is sensibly shorter than before the crisis and an adjustment of the cardiovascular system to the new circulatory regime has become necessary. This presupposes a cardiac wall capable of reacting efficiently and of suiting its activity to the variations of volume and blood pressure. This adjustment should be the especial function of the auricle.

If the cardiac wall is inert the adjustment does not take place and the first alternation of strong and weak systole is reproduced in the fashion of a sustained pendulum movement. No activity resident in the auricular wall occurs to check or annul this movement. The very regularity of the alternation is consistent with this conception.

The tracing in figure 53 shows how, after a premature systole, the pause which follows, permitting a better filling of the ventricle, seems to determine the alternation of the pulse, which reinforces each time the pause following each new premature systole.

The tracing in figure 146 shows how the phase of arterial compensation follows the phase of the variations of pressure, initiated by rhythmic troubles. This phase of compensation coincides with the crisis of tachyarrhythmia. Figure 143 shows a phenomenon of this kind, where the arterial adjustment is far from being perfect and is realized only at the close of the tachyarrhythmic crisis. On the tracing, figure 54, the adjustment occurs toward the end, while in the major portion there is alternation of the pulse and of the complexes. On the tracing, figure 140, the compensation occurs in the central portion, putting an end to the arterial alternation.

From the preceding data it seems that the alternation of the pulse is due to lack of compensatory activity of the cardiac walls and especially as the result of rhythmic troubles. This lack of activity of the auricles, passively undergoing the initial pendulum impulsion, entails a variation in their form—a variation equally pendular. It is with that variation of form, if it is marked, that I would associate the alternation of the electric complexes. It is well in this connection to recall how much the electric complex is modified solely by the respiratory movements and the changes which they cause in the form of the heart and great vessels. The tracing in figure 57 is remarkable because it shows us that a quite rapid respiratory rhythm has determined a change in the form of the complex once in every two times. In speaking of the influence of the respiratory movements on the complexes, in the tracings which we have previously given, the modifications of these complexes were shown on a much longer curve, and there occurred an alternation not only between two systoles deformed in different fashion, but between two series of systoles. The deformation was progressive in one direction for one series, and in a contrary direction for another series.

If the variations of intrathoracic pressure are sufficient to impose a variation of form on the heart and thus a variation of electric complex, one need not be surprised at changes due to unequal filling of the auricular cavities. The latter is caused by an initial and uncompensated disturbance of the ventricular rhythm, and is manifested by an analogous phenomenon. The inconstancy of the electrical alternation, its variable relation with the alternation of the pulse which harmonizes an ample complex with either a strong or a weak pulse, need cause no surprise. The alternation of the pulse and of the complexes is absent when the auricular tonicity is sufficient for compensation from the time of its appearance. On the other hand it will be seen when there is a myocardial alteration or overwork of the auricle laboring with a too rapid rhythm.

Change in Form of the Complexes in the Proiosystoles

In the premature systole or proiosystole (the extrasystole of various authors) we shall see that the ventricular complex may take a form very different from the normal. When the extrasystole is interpreted as a systole which is derived from an ectopic stimulus and which does not rejoin the specific system until after a more or less aberrant course, the anomalous morphology of the complex is explained by this same aberration. We believe, however, that the proiosystole differs from the normal only through its precocity. Its mechanism differs in no wise from that of the systole. If at times, but by no means always, the ventricular complex is deformed, it

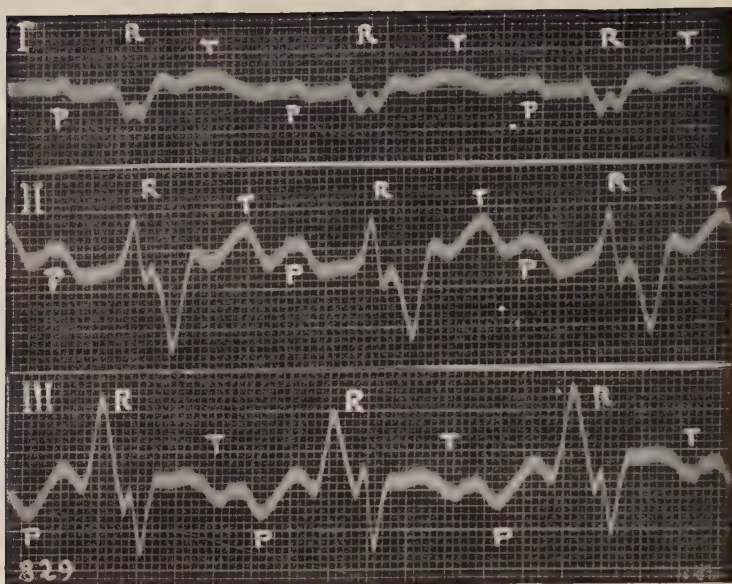


FIG. 58. Deformed ventricular complexes.

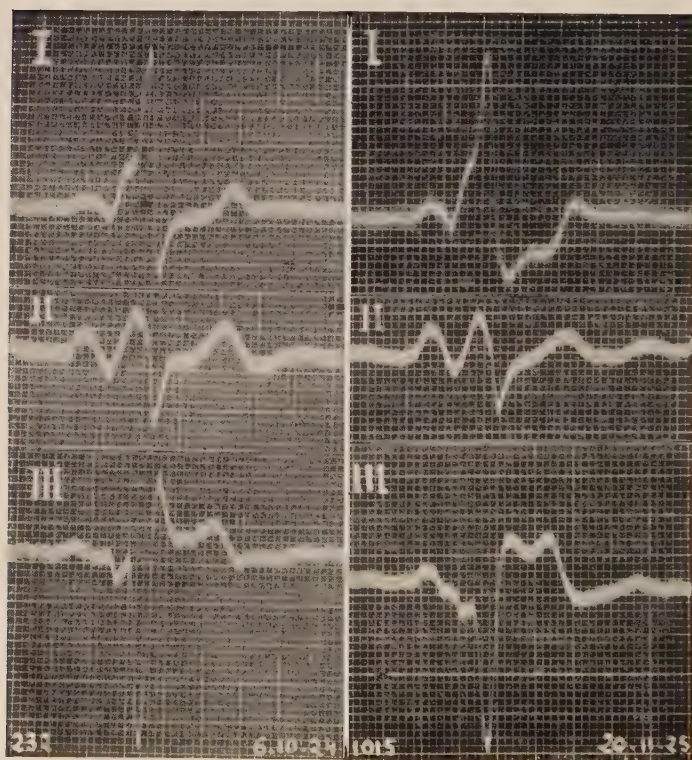


FIG. 59. Deformed ventricular complexes. These two tracings were taken from the same patient at an interval of thirteen and a half months; their resemblance is remarkable.

is for quite another reason than its difference in origin or genesis. In the chapter on proisystoles we shall evolve an interpretation of the deformation of the complex under similar circumstances.

Variations of the Event T. For some years past great significance has been attached to variations of the terminal T of the ventricular complex. It is often inverted in Lead III without seeming to be due to anything abnormal. On the other hand, an unfavorable prognostic significance has been associated with the inversion of T in Leads I and II. Experimentally, when the base of the ventricle is heated or the apex chilled, T is inverted. We would emphasize the following fact: The uncertainty of the exact significance of the event when it is normal, makes every attempt at explanation of its anomalies at least premature. We do not fail to recognize their prognostic value, which seems genuine, and which does not depend on the pathogenic interpretation which might be given. But this is a purely clinical subject which we have not at present under consideration.

Deformation of QRS. Certain ventricular complexes present change of form of the QRS event, of which the tracings in figures 58 and 59 give examples. The event R has its base widened and its ascending or descending branches are hooked. It is admitted that the excitation is blocked at one of the branches of the bundle of His. In consequence of this block, the excitation first reaches the ventricle on the side opposite the block, and after this the ventricle of the same side; there is therefore a precession of one ventricle over the other, and in the balance of the electrogram it is the weight of the ventricle first excited which dominates and deforms the total electrogram by its influence. This hypothesis is based on the experiments of Eppinger and Rothberger,³⁵ which were repeated by Lewis and others. In these tests the region where the right or left branch of the bundle passes was mutilated by section or compression.

Our objection to these experiments is that, despite their ingenuity, they are in reality very crude, for they involve at the same time the bundle of His and the adjacent myocardium. Moreover, one has only to try to follow a Hisian branch on a series of histological sections, to realize the uncertainty of being able to differentiate the specific from the myocardial tissue. One cannot be sure that one has touched the specific tissue. Furthermore it must be noted that "the alterations of the electrogram do not always appear after section, but only after a certain interval when the myocardium begins to be distended" (Fr. M. Smith). We should also add that often the electrogram ought to be interpreted, classically, as due to disturbance of the right branch, although in the majority of cases it is the left ventricle which is injured. Some workers have gone even further and have en-

deavored to associate a certain type of electrogram with a block of the arborizations of the bundle of His. Besides the widening of the base, the notches in the ascending and descending limbs of R would be multiple and the events of the electrogram of slight amplitude. Nothing is proved save that a like ventricular complex corresponds to a lesion of the fibers of Purkinje, and we make the most express reservations as to the possibility even of demonstrating anatomopathologically that this interpretation is well founded.

We do not believe that the cause of the deformation of the ventricular complex should be looked for in the disturbance of stimulus conduction in the specific system. In our opinion the part of the undulating line which constitutes a ventricular complex is composed of a series of points or "electrical vectors" each of which represents the algebraic sum of the electric variations produced at each of the successive moments into which the duration of a ventricular systole is decomposed. At each of these moments the form of the heart is different, so that the habitual form of the complex corresponds to the electric phenomena transmitted by a myocardium which passes through a series of differences in form. As long as these changes in form succeed one another in the same order and are reproduced with the same successive types, the electric phenomena present the same aspect; but if these changes are atypical or do not succeed one another in the same order, the aspect of the electric complex is modified.

The change of form of the myocardium occurs harmoniously in a way when the myocardium is normal and in particular when a normal circulation assures both a regular and integral functioning of the contractile network. It is necessary that all of the vascular territories should be governed by a well-balanced circulatory system, for with this equilibrium broken the changes in form of the myocardium become dysharmonic. We shall find this explanation again when we study the proiosystoles. It would be as well here, when we are considering the permanent changes in form of the ventricular complex, to interpret them as the result of a circulatory disturbance, and more particularly of one due to a permanent arterial lesion. There is really an actual claudication of the contractile myocardium which is translated by an anomaly of the complex, of which, however, the form alone is changed, its duration remaining unaltered.

The explanation proposed by Deglaude that these changes in form of the ventricular complex are due to fatigue of the myocardium, is not in contradiction with our own. In a recent article by Professor Clerc³⁶ and his collaborators, Drs. Bascouret and Lévy, there is a very complete list of the numerous works published on this subject.



FIG. 60

FIG. 60. Electrocardiogram and heart sounds. The first sound immediately follows the R wave. At the end of the second, or long, pause comes the P wave. P, R, and T mark certain phases of function of the cardiac chambers—phases with which the heart sounds and pauses are in relation.

FIG. 61. Sinus tachycardia, rate 142 per minute. The P wave remains between T and R. PR is short and equals 10/100 second.

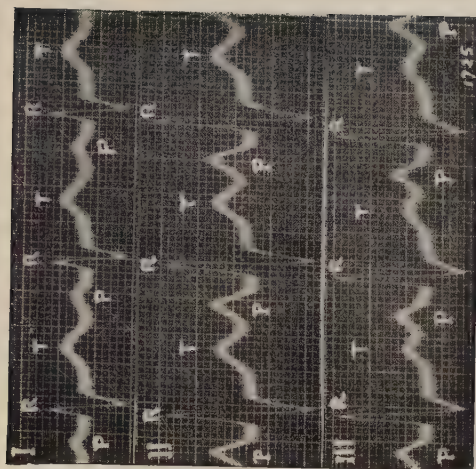


FIG. 61

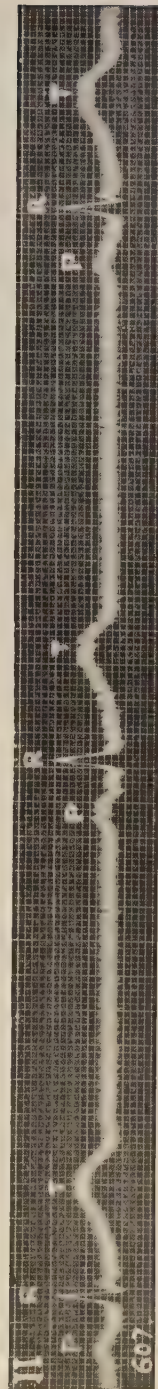


FIG. 62. Sinus bradycardia, rate 39 per minute. The PR interval is 16/100 of a second

What we have just written concerning the ventricular electrogram and the significance of the events which compose it and of their anomalies of form, shows that our knowledge on these points is incomplete and the conclusions already announced are controvertible. It seems evident, however, that the morphological anomalies of the auricular and ventricular electrograms correspond to something. It is on the side of the morphology of the complexes that most of the studies of electrocardiography are directed today, and this is without doubt the electrocardiography of the future.³⁷

In this work we are considering the electrocardiogram from quite a different point of view, and we retain only the two very precious landmarks which it furnishes us, namely P and R (see fig. 60). All authors are in accord as to their value for they are the signals: P indicating the beginning of the auricular contraction and R the beginning of the ventricular contraction. The phlebogram, in registering the volumetric variations of the external jugular vein, gives us analogous data, but the electrocardiogram has this double superiority of being very easy to obtain and to decipher, whatever may be the anomaly of the cardiac rhythm and whatever the efficiency of the myocardial contraction. Thanks to the signals P and R, which it gives us, we shall be instructed in regard to the different phases of functioning of the cardiac cavities and to the anomalies of this functioning. This is the real object of this work.

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PART II

THE ANOMALIES OF THE CARDIAC MECHANISM

CHAPTER V

THE ANOMALIES OF THE CARDIAC MECHANISM

So long as we were restricted to a study of the sound heart, it was possible to consider this organ as a whole with closely united parts and it seemed to be legitimate to speak of a cardiac rhythm. But we have been forced to recognize that the auricular and ventricular cavities may function independently of each other. There is then not one rhythm of the heart but two rhythms, auricular and ventricular.

For the notion of a cardiac rhythm it is preferable to substitute that of a cardiac mechanism, the elements of which we are going to enumerate. First the auricular and then the ventricular functioning should be taken up separately, and later the relationship between the two will be considered. The constituent elements of the cardiac mechanism are as follows:

1. The velocity of each rhythm which normally should be constant.
2. The dissociation between the auricular and ventricular systoles which should be such that the former precedes the latter, and precedes it by an interval comprised between certain limits.
3. The relation of the rapidities of the two rhythms, auricular and ventricular, which should be a relationship of equality.

HOW TO READ THE ELECTROCARDIOGRAM

Before approaching the study of the tracings reproduced in the following chapters, it will be necessary to consider the way in which an electrocardiogram should be read.

We may accept the classical hypothesis of a major sino-auricular center which dominates the entire heart, with the possibility that, under stress in certain pathological states, aid may come from the minor centers; or we may consider the other hypothesis, which is the one we maintain, of two centers always in function—one, the atrionector which commands the auricle, the other, the ventriculonector which commands the ventricle. According as one or the other of these theories is adopted all the facts observed in electrocardiography will assume different aspects, and more particularly we shall have to read the electrographic tracing in different ways.

According to the classical hypothesis there is an electrocardiogram which corresponds to the functioning of the heart considered as a whole and constituted by a succession of electric events, PQRSTU. In consequence

we must consider primarily the relations of these different events among themselves and the relations of the different groups PQRS^{TU} to analogous neighboring groups. The entire classification and all the nomenclature of classical electrocardiography rest upon what may be styled this unilinear reading.

According to the hypothesis of two cardioneotors, the reading should be bilinear. One must in thought separate the auricular from the ventricular electrocardiogram and consider at first the auricular and ventricular series individually. It is only secondarily that we shall be concerned with the relation which exists between the two series.

In the exposition which will follow of the anomalies of the cardiac mechanism, we shall therefore be obliged to take our stand successively at these two points of view. First we shall note how the facts are to be interpreted from the classical angle, and then from the new viewpoint which seems to us to approach more nearly to reality. When all the elements present in the cardiac mechanism are normal the mechanism is normal, but it becomes abnormal if any one of them is abnormal.

In reality none of the constituent elements of the cardiac mechanism has such a precise value that we can speak of anomaly either on one side or the other. There is no normal velocity and no normal dissociation, and we can speak only of averages. Thus it is that the frequency of the heart beat may oscillate between 60 and 80 beats a minute in hearts equally normal, the figures being only approximate. We often note a frequency below 60 in subjects who seem free from all cardiovascular damage. Likewise the frequency may increase to high figures as a result of effort or of emotion, without authorizing us to regard the cardiac mechanism as abnormal. It is not a question of anomaly properly speaking, but of greater or less activity.

We may reserve the terms bradycardia and tachycardia strictly to these differences in rapidity, although they may be inexact (figs. 61 and 62, p. 113). We admit that it is the entire heart which beats more slowly or rapidly, so that tachycardia or bradycardia results from the hyperactivity or hypoactivity of a single center, the sino-auricular node, whence the expression sinus tachycardia or sinus bradycardia. But this is only apparent, for in such cases it is really a question of the combination of two tachy-rhythmias or bradyrhythmias coupled, the one auricular, the other ventricular. This combination is the consequence of the hyper- or hypoactivity of two centers, the atrioneotor and the ventriculoneotor. If the variation, plus or minus, of activity be the same for each of the cardioneotors the rhythms of the corresponding cardiac cavities, whether increased or de-

creased, will remain equal. The relations of P and R remain the same and we get the impression that they are united, that it is the whole heart which is accelerated or retarded.

The activity of the cardioneurons may be augmented or diminished under the influence of different factors—nervous, humoral, toxic, or circulatory. It is well known from the researches of Weber, that a stimulus applied to the peripheral end of the pneumogastric causes a slowing of rhythm and that stimulation of the sympathetic causes an acceleration of the same. But we believe that very often the circulatory factor intervenes in a predominant fashion, and it may even be perhaps by the intermediary of the circulation that the nervous action is exerted. An augmentation in the delivery of the artery of the atrionector will increase the frequency of the auricular rhythm, and the same is true of the ventriculonector and the ventricle. Conversely, slowing of these rates may be the result of a lessened delivery by the arteries of the cardioneurons. In order that the variations of the two rhythms may remain equal, the arterial supply to the two cardioneurons must vary in equal degree. This is the case when the entire coronary system participates in the variation, and not only a single artery. The heart as a whole seems to obey only one center, but in reality it obeys two which are subjected to equal influences. We may be able to show that the impression which one has of a heart, accelerated or slowed as a whole, is inexact. If the cardiogram be examined closely it will be seen that the interval PR is not a fixed one, but varies inversely with the frequency, diminishing when that increases, lengthening when it decreases. The curves reproduced below (figs. 63, 64, 65) which show this variation were obtained from a subject, in whose circulation the activity had been modified by intravascular injection of a mixture of adrenalin and nitroglycerin.

The variation in the PR interval is clearly explained by the fact, if it is a fact, that the production of P and R is in direct relation to the wave of blood propagated in the arteries of the atrionector and ventriculonector respectively. In experiments made in collaboration with Dr. Luisada¹ a fact has been established which seems to confirm this hypothesis.

We measure the interval which separates the arrival of the wave of blood to the arm and to the leg respectively. This interval is designated by HT, H marking the delivery of the brachial (humeral) and T that of the tibial wave. HT varies in inverse ratio to the frequency.

If we admit that the event P marks the moment when the arterial wave arrives at the atrionector, and R the passage of the wave to the ventriculonector, then the interval PR measures the interval between the arrivals of the two waves. Now PR behaves like HT and we may compare the arteries of

FIG. 63



FIG. 64

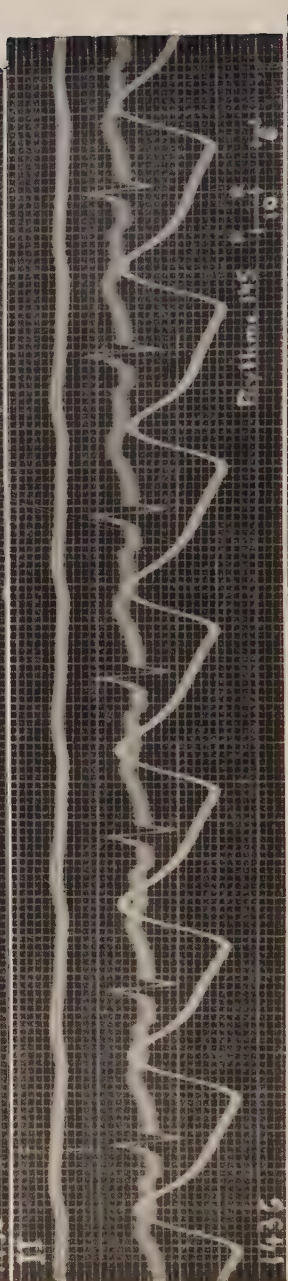


FIG. 65



FIGS. 63, 64 AND 65. Experimental tachycardia. When the rhythm accelerates, the intervals PR and HT diminish, to lengthen again when the rate slows. These three tracings were taken at different times in the experiment, but belong to the same film.

the extremities and those of the cardioneectors, since in each couple there is a short artery, the brachial or that of the atrioneector, and a long artery, the tibial, or that of the ventriculoneector. If this hypothesis is true the variation of PR and of HT in inverse ratio to the frequency is readily explained and is nothing more than a question of mathematics. The waves propelled from the heart to the periphery are like trains leaving the same point. Suppose they go at a speed of 50 kilometers an hour; that one which reaches a station 50 kilometers from the point of departure arrives at the end of an hour, while another leaving at the same time takes two hours to reach a station 100 kilometers away. The interval between the two arrivals, PR or HT, will be one hour. If the speed of the two trains is 100 kilometers, the interval between the two arrivals will be a half hour. The interval PR or HT, therefore, varies in inverse ratio to the speed.

Whether or not this hypothesis is well founded, in any case P and R are clearly independent of each other. One might perhaps strictly explain, according to the classical theory, this diminution of PR when the frequency increases, by saying that the conductivity of the bundle of His is more rapid because the heart beat is more rapid and that consequently PR must diminish. But we shall see other facts later in which an acceleration of rhythm is accompanied by an elongation of the PR interval. This, some say, occurs because the conductivity of the bundle of His is slowed, owing to overwork from acceleration of the heart beat. We cannot admit that in one case the energy, and in another the fatigue, of this bundle can be invoked to make the observed facts agree with an hypothesis. If, on the other hand, it is a question of two centers, one dominating the auricle and the other the ventricle, we can imagine that the activities of these centers may be differently excited and as a result the electrical events which signal their entrance into mutual activity will be caused by different factors. We have observed the same facts experimentally by simply utilizing the action of the amyl nitrite by inhalation.

The length of the complexes varies as PR and therefore is in inverse ratio to the rapidity. If we compare the tracings shown in figures 63, 64, and 65, we find the following measurements: the ventricular complex RST measures respectively 32, 24, and 30 hundredths of a second when the rate is 78, 145, and 120. In the chapter on discordant ventricular bradyrhythmia we shall find this variation of the ventricular complex still more marked (fig. 162).

It is probable that the auricular complex varies in the same way, but as it is covered in part by the ventricular complex all measurement is impossible. However, we may cite the cases in which, as a result of unequal rapidity of

the rhythms, the auricular complex is quite unmasked. We then see that the complex diminishes in length when the auricular rate became more rapid. (Compare the tracings which we give in the chapter on auricular tachyrythmia.) The regularity in rapidity of action of the normal cardiac mechanism is only approximate, for when we measure the interval between the successive auricular or ventricular contractions we note slight variations of a few hundredths of a second. There is nothing mathematically exact in this.

The difference in time between the auricular and the ventricular systole varies, oscillating between ten and fifteen hundredths of a second, although certain authors make the limits much wider. Thus Schrumpf states that it may be lowered to five hundredths of a second and Lewis that it may be raised to twenty-one hundredths of a second, without such extreme values indicating a pathological state. The interval PR measures this difference. It is either the distance between the summits P and R, or that between the feet of these events, whence it results that in reality one sometimes does not measure PR but PQ. This measurement is, however, rather rough because the form of P varies with the individual and even in the same person at different times, according to the lead and the existing conditions.

Granting these preliminary reservations, when any one of the elements which go to make up the cardiac mechanism becomes abnormal one may correctly speak of an anomaly of the cardiac mechanism.

Every important modification of a single element may bring about a modification of one or the other elements of the cardiac mechanism. Thus if the condition of the equality of the rhythms is not fulfilled that of the mean value of the time relation ceases to be valid. Several conditions may be lacking at the same time and all combinations among these deficiencies may be seen. But to be clear each of the constituent elements of the cardiac mechanism should, so far as possible, be studied separately.

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CHAPTER VI

ANOMALIES CHARACTERIZED BY VARIATION OF THE RHYTHMIC FREQUENCY

In the normal heart the auricular contractions succeed one another at equal intervals and this is also true of the ventricular contractions. One may consequently foresee the point where upon a tracing an auricular or ventricular contraction should appear. The impression of a rhythm is very clear.

On the other hand in the clinic one often notes that the auricular or the ventricular systole does not appear at the moment foreseen—that it is anticipated or retarded. We call the systole which is premature the proiosystole, from *πρώιος*, premature; and the one which is retarded the hysterosystole, from *ὑστέρος*, retarded.¹

Let us study in succession the two types of systole.

THE PROIOSYSTOLES

Before describing the proiosystoles it will be useful to explain, as briefly as possible, what is understood classically by extrasystoles. The phenomenon described under that name comes in fact within the group of the proiosystoles.

The Extrasystoles

An extrasystole is a premature or ectopic systole. Premature, because it comes before the moment when the rhythm of the previous beats would lead us to expect it. This character of prematurity, which is never wanting, is a fact which we can only state without explaining.

According to Lewis the extrasystole is ectopic, while Hering calls it heterotopic. This character is hypothetical and it is according to this purely hypothetical character that the extrasystoles are classified. The normal systole is the result of a stimulus which originates in the sino-auricular node—a “normotopic” stimulus. The extrasystole is the result of a stimulus which originates somewhere outside of the sino-auricular node, a “heterotopic” stimulus. Extrasystoles of different type correspond to the different centers in which they are presumed to originate. Let us first define the two expressions “post-systolic pause” and “post-extrasystolic pause.” It is the interval which separates respectively a normal systole or an extrasystole from the succeeding normal systole.

Following the classical theory there are four principal kinds of extrasystoles (fig. 66):

1. Sinus extrasystoles.
2. Auricular extrasystoles.
3. Atrioventricular [or junctional] extrasystoles.
4. Ventricular extrasystoles.

The *sinus extrasystole* is a premature systole of the entire heart, auricle and ventricle. The auricular and ventricular events are both dislocated, as it were, up-stream; PR is unmodified. The complexes have a normal form. The post-extrasystolic pause is slightly less than the systolic pause.

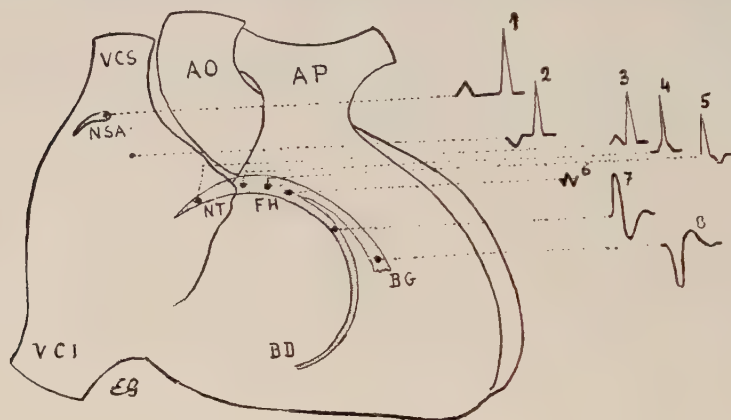


FIG. 66. Form and assumed seat of the principal types of extrasystoles. Schematic heart, right side facing the reader. VCS, VCI, superior and inferior venae cavae; AO, aorta; AP, pulmonary artery; NSA, sino-auricular node; NT, Tawara's node; FH, bundle of His and its right and left branches, BD and BG. 1, The normal cardiac systole; the sinus extrasystole starts from a point very near the normal, and differs only as a form of the latter. 2, Auricular extrasystole. 3, 4, 5, Atrioventricular extrasystoles in their three varieties—high, medium, and low. 6, 7, 8, Ventricular extrasystoles with their three varieties—central bigram (6), dextrogram or basal variety (7), and levogram or apical variety (8).

The *auricular extrasystole* differs from the former only in the fact that the post-extrasystolic pause is a little greater than the post-systolic. PR is at times slightly diminished, and the auricular complex may be reversed.

The *atrioventricular extrasystole* is a premature systole of the entire heart, but the relation of the auricular and ventricular events is modified: PR is notably diminished, absent or negative. The auricular complex may remain normal or be inverted. The ventricular complex has its normal form. The post-extrasystolic pause is lengthened and at times compensatory—that is to say, it compensates exactly by its elongation for the

shortening of the interval between the extrasystole and the normal systole which preceded it. According as PR is diminished, absent, or negative the atrioventricular extrasystole is called supranodal, nodal, or infranodal.

The *ventricular extrasystole* is a premature systole of the ventricle alone. The auricular systole occurs on time. The ventricular complex is habitually deformed. The post-extrasystolic pause is compensatory.

The deformation of the ventricular complex occurs in the three types described by Kraus and Nicolai as A, B, and C.

Type A represents a negative followed by a positive event. It is designated by the letter A, the initial letter of the word *Apex* because it has been observed experimentally when the region of the apex is excited. Type B consists of a positive followed by a negative event. B is the initial letter of the word *Basis*, this type being one which responds to an excitation in the region of the base. Type C is a complex habitually triphasic, of medium amplitude. C is the initial of the word *Centrum*.

Type C approximates closely the normal ventricular complex but A and B differ from the latter notably and have a very marked biphasic aspect, the positive and negative phases following each other without the interposition of an iso-electric line. The terminal phase which approximates the T of the normal complex is always of wide amplitude. It follows from this, even in cases where the beginning of the extrasystolic complex differs but little from that of the normal complex, that the very apparent terminal phase of the extrasystole affects the tracing. When positive it makes a great ridge; when negative it depresses the curve strongly and marks the extrasystole.

The extrasystolic complex, despite its distortion, has a length equal to that of the normal complex. This fact is of service for the analysis of curves which are slightly complex. Like the normal complex, the form of the extrasystolic complex varies with the lead. It is by convention that Lead II has been chosen and that A and B are characterized by the positive or negative direction of the initial phase of the complex in that lead.

In comparing the complexes we find in the three leads the same varieties described in connection with the normal complex. In general, but not always, the events have the opposite sign in Leads I and II, the same sign in Leads II and III. Type A is often considered (although this is not strictly correct) as an extrasystole which proceeds from the left heart, as a "levo-gram"; type B which comes from the right heart as a "dextrogram," and type C as representing an extrasystole which was born in the septum between the two hearts.

We will add that Hoffmann employs a different nomenclature and con-

siders the complex separately in each of the Leads, I, II, and III. He calls type A the complex with initial positive phase, type B the complex with initial negative phase, and type C the polyphasic complex. But there is here a source of regrettable confusion, for type A in Lead II of Hoffmann corresponds to type B of Kraus and Nicolai, and *vice versa*.

One form of ventricular extrasystole may exhibit a complex of normal aspect, namely the Hisian variety. Another variety, called dissociative (*décalante*), is characterized by the fact that the post-extrasystolic pause is equal to the post-systolic. This variety is seen when the rhythm is slow.

In addition to the four principal forms of extrasystoles which we have just reviewed, other secondary forms have been described, called interpolated retrograde, and blocked auricular.

The *interpolated extrasystole* is a premature ventricular systole occupying the space TP, and of such character that the interval between the DP groupings, which precede and follow it, is equal to the usual interval. The insertion of the interpolated extrasystole has therefore brought no change into the rhythm of the normal systoles. One notes at times an elongation of PR in the group which follows the extrasystole. The interpolated extrasystole is observed with a general rhythm of the slowed heart.

The *retrograde extrasystole* is a premature systole of the heart, characterized by a premature and deformed ventricular complex, and followed by a premature and inverted auricular complex.

The *blocked auricular extrasystole* of Hewlett² is a premature auricular systole, not followed by a ventricular complex.

A patient shows in general always the same type of extrasystole, and tracings taken after an interval of several years have the same physiognomy. On the other hand a subject may present on the same tracing extrasystoles belonging to different varieties. Extrasystoles are seen as single events or few in number—or as multiple events which reappear regularly or irregularly. They may be grouped in actual volleys of varied length, interrupting the normal rhythm.

Explanation of the Extrasystoles

The hypothesis which has served as the basis for a classification of extrasystoles, is, as already stated, as follows: The extrasystole is a contraction of ectopic origin. Oddly enough, however, it is admitted that the sinusal extrasystole originates in the normal sino-auricular center, its sole characteristic being unseasonableness. All the other varieties are at the same time premature and of ectopic origin.

The seat of the anomalous excitation is the myocardium of the auricle in

the auricular extrasystole, the atrioventricular or nodal center in the extrasystole of that name, and some point in the bundle of His or of one of its branches in the different types of the ventricular systole.

This hypothesis is based on experimental facts. In stimulating different regions of the heart by means of an electric current, premature contractions have been obtained which reproduce the different types of extrasystole. It is admitted that the stimulus applied to the heart always gains the specific system and then, following the latter, reaches the contractile myocardium.

The sinus extrasystole proceeds from a stimulus which, originating in the sino-auricular node, determines the auricular and ventricular contractions in succession, just as does the normal stimulus when produced at its normal time.

The auricular extrasystole is caused by a stimulus, originating in the myocardium of the auricle, which sets off the auricular and ventricular contractions. The normal sinus stimulus which follows this can no longer act because it intervenes only after a certain delay and then finds a myocardium discharged by the ectopic stimulus.

The atrioventricular or nodal extrasystole is the result of a stimulus originating in the region of the node of Tawara. It causes the contraction of the auricle which it approaches from below upward and the contraction of the ventricle which, as in normal conditions, it approaches from above downward. From its center of origin, the stimulus, radiating in the two directions, reaches auricle and ventricle at times which vary with their respective distances from the ectopic center. The cardiac cavity first reached is the first to contract; it may be auricle or ventricle according to the situation of the ectopic stimulus. The normal sinus stimulus is unable to excite contraction in a myocardium already discharged by the stimulus propagated in the retrograde direction. The ventricular complex, as in the preceding types, is normal, because the ventricle is urged to contract by a stimulus which reaches it by the normal route. When it is abnormal we must suppose that the extrasystolic phenomenon is complicated.

The Hisian extrasystole is the result of a stimulus which originates in the bundle of His before its bifurcation. The stimulus, following down the specific system, reaches the ventricles by the normal route. The complex remains normal.

The ventricular extrasystole comes from a stimulus which originates in one of the branches of the bundle of His, and reaches normally only the corresponding ventricle. It must make a *détour* ("aberrance" of Lewis) to arrive at the other ventricle. The ventricular complex is deformed.

In the case of the ventricular or Hisian extrasystole, the normal sinus

stimulus no longer acts upon the ventricle. It arrives in fact after the ectopic stimulus and finds a myocardium already discharged and therefore refractory. The auricle alone responds to the normal sinus stimulus.

On the other hand the ectopic ventricular stimulus ascends, indeed, toward the auricle, but does not act upon it because the auricle has been discharged by the normal stimulus.

This explanation does not hold good in the case of an interpolated extrasystole. The post-extrasystolic pause is in fact amply sufficient for the abnormal stimulus, ascending toward the auricle, to find it ready again for contraction. Yet this does not occur. "The reason why many of these extrasystoles do not pass to the auricle and produce a retrograde contraction remains a mystery" says Lewis. "In certain cases," he adds, "one may admit that the retrograde contraction would coincide with the next normal auricular excitation, but this is not always the case."

The blocked auricular extrasystole of Hewlett is believed to have the same origin as the auricular systole, that is to say, to proceed from a stimulus originating in the auricular myocardium. But this stimulus, after having provoked the contraction of the auricle, is unable to provoke that of the ventricle because it is blocked at the level of the bundle of His. It is in fact impossible to attribute this to a refractory state of the ventricle.

To sum up, there is an extrasystole because one portion of the heart, other than the major sino-auricular center which presides over normal systoles, usurps the command of the entire heart or of one of its cavities.

It remains to explain why the major center is dispossessed by the ectopic center. This may happen in two ways. In certain cases the ectopic stimulus precedes the normal stimulus, either because it acts with greater rapidity or because it reaches a certain part of the heart first. This part, discharged by the stimulus which is the first to arrive, no longer reacts to the delayed stimulus. In other cases, the normal stimulus is prevented from acting because a part of the myocardium behaves as if excluded from its domain as a result of troubles of conductivity. This region of the myocardium, withdrawn from the action of the normal stimulus, responds to an autochthonous stimulus which is no longer in competition with the normal of higher frequency. Two rhythms may thus coexist, that of the excluded zone and that of the remainder of the heart.

Kaufmann and Rothberger³ combine the two previous ideas in the building up of their theory of the *parasystole*: The increased rhythm of activity and the exclusion of one portion of the myocardium. They hold that one region of the myocardium is excluded and also has a rhythm of activity more rapid than that of the major center. This more rapid rhythm should give it

command over the entire heart, but its exclusion prevents it from exerting any such influence. The difficulty of conduction at the periphery of the excluded zone protects the rest of the heart. There is a protective blockade (Schutzblockade). Further this block varies in completeness; it is greatest when the vagus is predominant and least when the sympathetic prevails.

The extrasystole results from the momentary rupture of the block, an event which may be reproduced at variable intervals, regular or not. One or several extrasystoles may be interpolated into the rhythm of the rest of the heart, with the well known modalities of bigeminism, of polygeminism, or of extrasystolic volleys. A complete rupture of the block will bring about a crisis of tachyrrhythmia, which will cease only with the reconstitution of the barrier of poorly conducting tissue.

We might thus explain a peculiarity often noted, namely, that the interval between isolated extrasystoles is a multiple of the interval between two extrasystoles in a period of tachyrrhythmia. All this is as if alongside of a sinus rhythm there were a concealed rhythm, ordinarily surrounded in one part of the heart by a safety blockade, which may for a moment be broken through, whence the invasion of the heart by an unblocked rhythm. From then on this unblocked rhythm and the sinusal rhythm interfere with each other. The contractions dominated momentarily by the unblocked rhythm would merit the name of parasystoles, the systoles being dominated by the sinus rhythm.

Lewis combines the notion of the exclusion of a zone of myocardium, more particularly that of the ventricle, with the hypothesis of the "circus movement" and seeks thereby to explain at the same time the ventricular extrasystoles and the ventricular tachyrrhythmias.

Finally de Boer⁴ applies to the extrasystole his theory of the fractionated systole which will be studied in the chapter on the anisorhythmias due to discordant auricular tachyrrhythmia.

All of the preceding theories admit in short that the extrasystole results from an anomalous ectopic stimulus which would be diffused through the entire heart and dominate the same, if its action were not restricted by that of the normal sinus stimulus which persists and which also spreads throughout the entire heart.

The heart is thus divided into two domains in each of which the stimulus which is first to arrive dominates.

Critique of the Classical Theory of the Extrasystoles

We do not accept the classical explanation of the extrasystole which we have just set forth. The phenomenon of the extrasystole has two charac-

teristics, prematurity and ectopia. The first of these is unquestionable, it is an actuality, but the second is hypothetical. In favor of the hypothesis it is claimed that it is supported by the result of experiment. Furthermore the hypothesis seems to be confirmed by the fact that the extrasystole is a deformed complex. Let us examine these two ideas.

Experiments made on the hearts of animals, most frequently of the dog, seem convincing. A stimulus applied to a different portion of the heart causes extrasystoles and these are of different types. It is necessary to call attention first to the fact that the results of experiments, those on the dog for example, do not have the fixity which would appear from the published scheme. Hoffmann in particular has insisted on the variability of the results.

On the other hand, when we pass from the animal to man, the results are no longer the same. Hoffmann struck light blows of the percussion hammer all over the accessible surface of the heart in a man who was undergoing a resection of the third to the tenth ribs. Whatever the point of accessible surface excited he obtained invariably in the first lead type B and in the second and third leads the type A. He was never able to obtain type A in Lead I, which would mean theoretically that he constantly tapped the left and never the right ventricle. This, as Hoffmann says, is unlikely.

On the other hand it is remarkable, if we examine the experimental tracings given by Lewis, for example, where the moment of excitation is recorded, to note that the extrasystole is often not produced until after several repetitions of the stimulus.

The extrasystole presents this constant and curious peculiarity that the duration of the extrasystolic ventricular complex, which alone is measurable, is exactly equal to that of the systolic complex. This statement agrees poorly with ectopia, which is defined as variable. The "aberrance" being variable, it is hard to understand how the extrasystole can nevertheless always develop at the same time.

All seems to happen as it would if the cardiac mechanism developed always in the same way and in the same length of time, the anomalous excitation intervening only to set this mechanism going sooner, but without any modification of its phases.

I would add that in the spontaneous extrasystoles in man the types are not numerous, that the same subject always presents the same type even after an interval of years, and finally that the ventricular extrasystoles, which according to the theory should be attributable to an abnormal excitation of the right ventricle, are the most frequent, although we know that it is the left ventricle which is the most often concerned pathologically.

The distortion of the extrasystolic ventricular complex seems to involve the idea of an ectopy of the systole; but the deformity exists only in certain extrasystoles and not in all. We have seen that there are hardly any but "ventricular extrasystoles" which are deformed, yet very many of these are not deformed. Distortion, then, is not a characteristic of the premature systole.

On the other hand there may be deformity when the systole is not premature but occurs on time. The tracing in figure 67 seems to me to merit reproduction in its entirety because of its rarity. Here we see a ventricular complex which is deformed but not premature. Figure 68 shows a series of ventricular complexes which are deformed and variously deformed, with occasionally regular alternation of two different types, but which cannot be regarded as always proisystolic complexes.

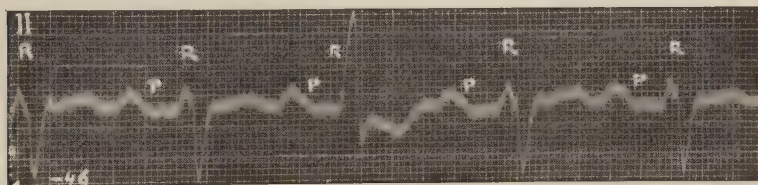


FIG. 67. Ventricular electrogram of extrasystolic, but not premature type. The five ventricular waves, R, are equidistant, and the same is true of the auricular waves, P. The third ventricular electrogram is of the type of a dextrogram and yet is not premature.

Figure 69 is of interest because it shows the ventricular pattern broken by various series of systoles closer together and grouped by twos, threes, fours, or fives. In one group of four, although this group does not differ in its arrangement from the others, two systoles in every four are marked by deformed complexes. It is difficult to put these in a class by themselves and to regard this deformity as signifying that they may have had a different genesis from that of the neighboring systoles.

From the preceding criticism it seems to us that the hypothetical character given to the extrasystole, to wit, that of being ectopic, is far from proven. The fact remains then that there are premature systoles, whence the term proisystole proposed by us as a substitute for extrasystole. This recalls the sole, constant specific character of the phenomenon which we are studying, namely its prematurity. For the rest it corresponds absolutely to the English expression, "premature beat."

What notion may we form of the proisystole? This is purely and simply a premature systole which differs from the normal only because it

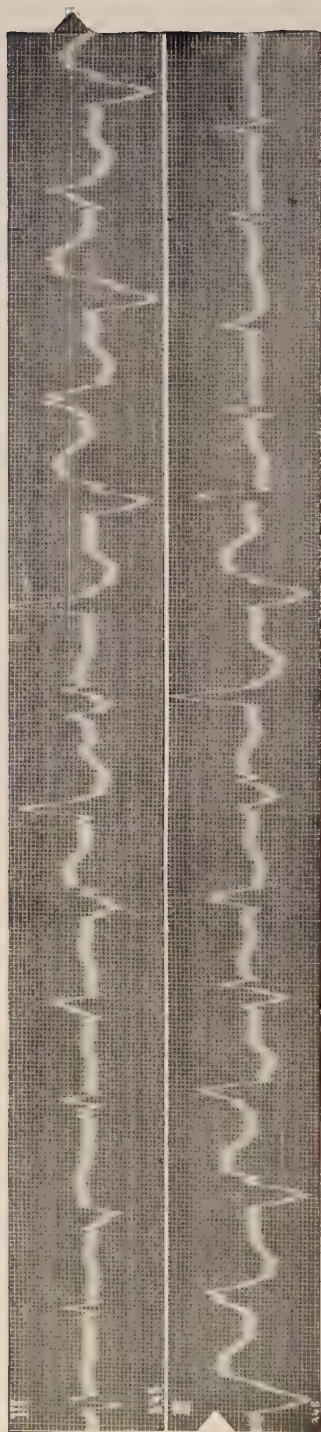


FIG. 68. Ventricular electrograms of the extrasystolic type, but not premature. The electrograms in this tracing are of quite varied form with a tendency to alternation of the two types, ascending and descending. Certain of these electrograms have a superficial resemblance to extrasystoles; but although the intervals separating the events are not very regular, certain among them cannot be regarded as distinctly shorter than the others, as is the case in extrasystoles.

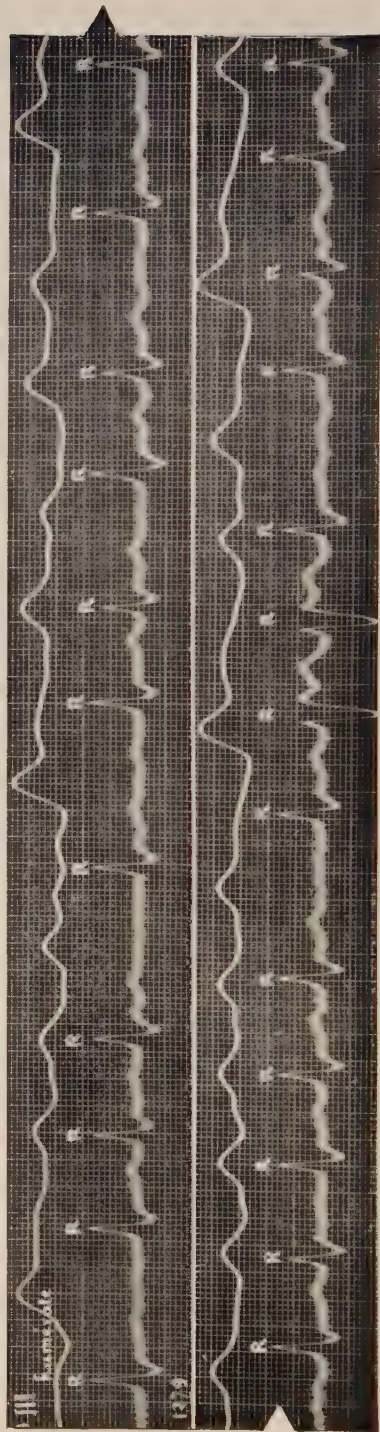


FIG. 69. Ventricular electrograms of extrasystolic type, but not premature

takes place sooner than is expected. Like the normal systole it is dominated by the cardionector and no other center comes into play. There is no ectopia but the cardionector enters too soon into its active phase and the cardiac cavity contracts ahead of time. There is nothing more to the proiosystole than this.

But since there is a systole of the auricle and a systole of the ventricle, each of these being related to the activity of the corresponding cardionector, there is a proiosystole of the auricle and one of the ventricle as well. These proiosystoles enter into combinations, either among themselves or with ordinary auricular and ventricular systoles, and all possible groupings result. The explanation of the proiosystole is therefore reduced to that of prematurity of the systole. Why is a systole of this or that cardiac cavity premature? Or, what comes to the same thing, why should the auricular or ventricular rhythm undergo this abrupt acceleration? The same problem obtains in the tachyrythmias and one may consider the proiosystole as an abortive tachyrythmia, or the latter as a succession of proiosystoles.

It is only necessary for the activity of a cardionector to be suddenly accelerated in order that a premature systole of the corresponding cardiac cavity may take place. We find here the conditions which we always invoke under the same circumstances—nervous, humoral, and toxic influences, often called into action by the intermediation of the circulatory system. It is the variation in the circulatory system which always dominates the mechanism of the heart, and the idea that one forms of the exact function of the cardionectors does not in any way affect the importance of changes in the circulation.

One may range oneself among the neurologists and regard the cardionector, at least for the time being, as the tract of union between the nervous system and the contractile myocardium. Or one may, with the myogenists, refuse to recognize any influence whatever on the part of the nervous system, and look upon the cardionector as the principal place for the production of a hormone or other stimulus necessary to start a systole. But whatever may be the rôle attributed to the cardionector, it is certain that it dominates the contraction of the corresponding cardiac cavity, and that its activity is strictly subordinated to its blood circulation. This may be modified in the way of an increase in the arterial delivery. Then a proiosystole will result from the sudden acceleration of the arterial supply. This is the hypothesis which we first proposed,⁵ but it does not agree with certain facts and in particular with the fact that in ventricular bradyrythmia, which, as we shall see, is associated with a decreased delivery of the artery

of the ventriculonector, proiosystoles frequently occur. But it is difficult to imagine that a stenosed artery may augment its output abruptly and in sufficient volume to produce this effect. Another fact likewise contradicts this first hypothesis. In a case of permanent auricular hysterosystole reported later, in which there was a stenosis of the artery of the atrionector, I noted on one occasion the occurrence of an auricular tachyrhythmia. This is the same phenomenon, only transposed from the ventriculonector to the atrionector. Here as there, it is difficult to reconcile the notion of a permanent diminution of the blood supply with a sudden and momentary increase.

I believe that this first hypothesis should be broadened and that it is more consistent to admit that the variation of the circulation, which acts to modify the activity of the cardionector, may depend sometimes on a change in the venous outflow. The proiosystole may therefore depend at one time on passive hyperemia due to a stasis of the venous blood through defective evacuation, and at another time on active hyperemia from augmentation of the quantity of arterial blood. We shall develop this idea further when we study the tachyrhythmias.

An alteration in the amount of blood in the cardiac center appears positively to be the cause of the proiosystole. This variation may occur in one or the other cardionector or in both at the same time. Their activity will therefore be modified individually or jointly. There will be an auricular, ventricular, or double (auriculo-ventricular) proiosystole. These proiosystoles, in combining differently among themselves, will give all the varieties of grouping which we found in the "extrasystoles" and certain other varieties which are not apparent in the usual classification.

Certain proiosystoles, not all, are as we have seen associated with a deformed complex and we interpret this deformity as due to the fact that the premature systole occurs at a moment when the myocardium is not normally relaxed. The ventricle in particular, the cavity most often interested, is surprised *in flagrante delictu* by the activity of the cardionector affecting it again and prematurely, and its new contraction will not be "harmonious." It will especially be inharmonious if the auricle, not involved contracts on time and in complete disaccord with the ventricle.

The events of the electrocardiographic curve which translate the electrical variations will be modified either in their genesis or in their algebraic sum, or more probably in their exteriorization. The electrical variations may in fact not differ in the premature and in the normal systole, if, as we think, they depend only on the beginning of activity of the cardionectors. But they are "seen through" a myocardial mass the form of which varies accord-

ing as the systole occurs on time or prematurely. The constancy of the length of the complex, whether or not deformed, is consistent with this way of comprehending the facts.

If our conception of the proiosystoles is correct, we shall have to consider separately the succession of auricular and ventricular contractions, the normal as well as the premature. It is necessary, in other words, always to halve the electrocardiac tracing mentally and read it on one hand as auricular, and on the other as ventricular. It is an error of interpretation to try to group this auricular with that ventricular event and thus to create combinations which do not correspond to anything real. On the other hand the deformity of the complex does not signify that the corresponding systole should be classed in a category other than that of the systole with normal complex. The important fact is that the systole is a premature systole, a proiosystole. It is always an indication of a sudden change in the activity of a cardionector—and nothing more.

That we may obtain proiosystoles of different types by experimental stimulation very probably depends on the fact that we thereby stimulate the activity, sometimes of one, sometimes of the other of the cardionectors.

We shall now proceed to study successively the auricular and the ventricular proiosystoles, either one of the two rhythmic series being liable to be interrupted by the proiosystolic accident.

Auricular Proiosystoles

The auricular series is rarely broken by itself. We have hardly ever seen this phenomenon save at the confines of a crisis of auricular tachy-rhythmia, either at the beginning or end. In the tracing reproduced in fig. 70 it is seen that the auricular series is broken twice by two proiosystoles, while the ventricular series is broken by only a single proiosystole. All of these premature systoles constitute in reality the onset of a crisis of tachy-rhythmia which has aborted. Then further on the crisis develops completely. In a second tracing (fig. 71) we see at the end of a tachyrhythmic paroxysm a systole-proiosystole couple break once again the ventricular rhythm and twice the auricular rhythm. The last auricular proiosystole thus remains isolated; there is on this second occasion no appearance of grouping of it with a neighboring ventricular proiosystole as was the case the first time.

The tracing shown in figure 72 is exceptional and its interpretation is difficult. We might see here an auricular rhythm broken by several proiosystoles while the ventricular rhythm remains unchanged. But we present this explanation with the greatest reserve.

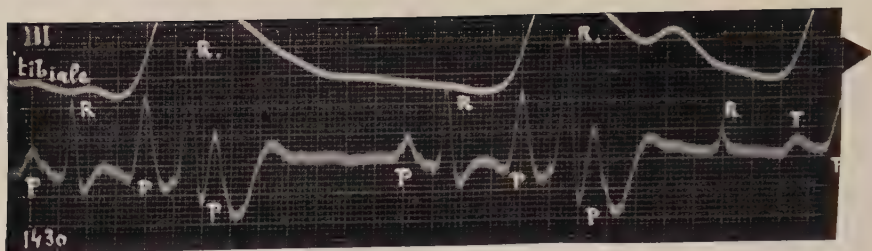


FIG. 70. A crisis of auricular tachyrrhythmia with its prelude. Two groups of ventricular and auricular proisystoles precede a paroxysm of auricular tachyrrhythmia. In each group there is a supernumerary auricular proisystole.

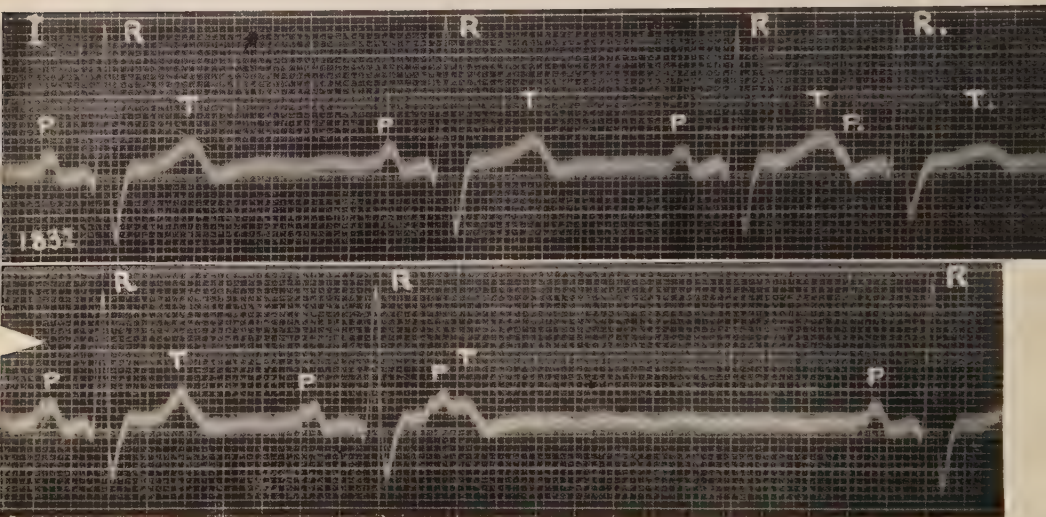


FIG. 71. The sequel of an attack of auricular and ventricular tachyrrhythmia. The attack itself does not figure in the tracing. *Ventricular series:* The ventricular rhythm after becoming normal is broken by a single additional proisystole, R₄. *Auricular series:* The auricular rhythm, after becoming normal is broken twice by a proisystole, P₄ and P₇. P₄ and R₄ may be considered as forming one group, called "auricular extra-systole," but P₇ is single and supernumerary.



FIG. 72. The events marked P? are difficult of interpretation, but probably correspond to auricular proisystoles

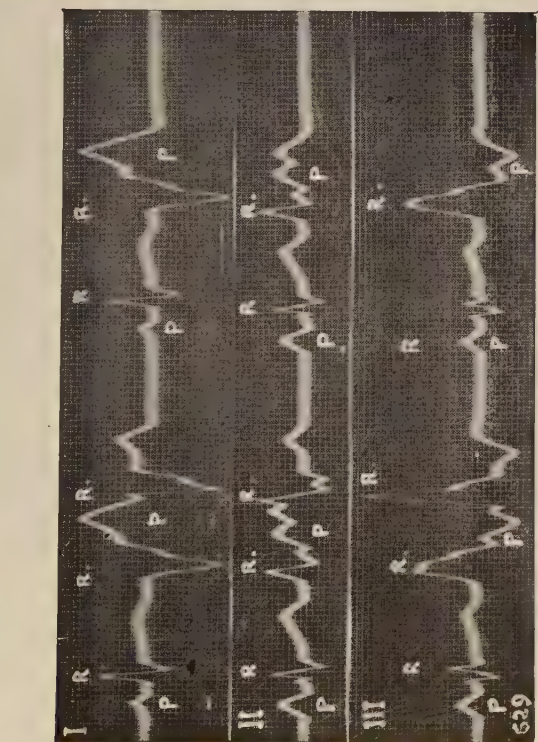


FIG. 73

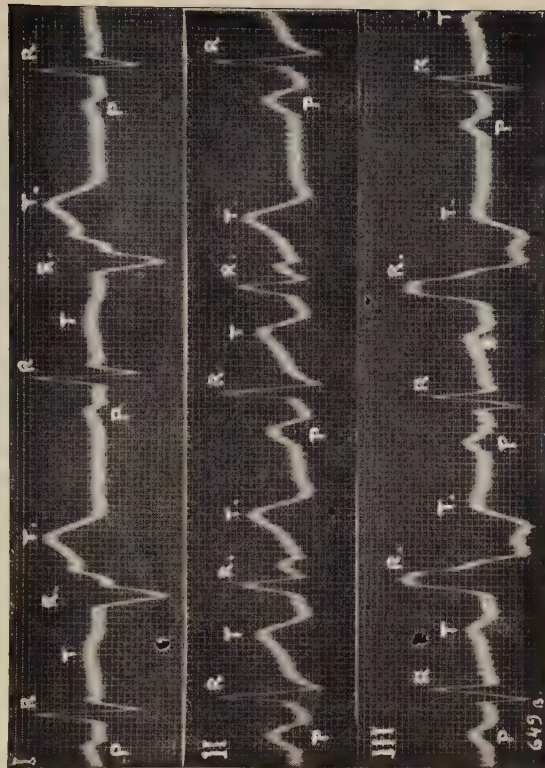


FIG. 74

FIGS. 73 AND 74. Reconstruction of the auricular series in tracings showing ventricular proisystoles. All these tracings are from the same subject. The registering of all the P waves of the auricular series is possible thanks to Lead II; in Leads I and III certain of the P waves are indistinguishable in the ventricular electrogram, but their presence is betrayed by certain irregularities in the curve which comparison with Lead II enables one to attribute to them. If it were not for Lead II, however, the identification of all the P waves would be very difficult and always doubtful. If we compared the end of the tracings in the two figures where a ventricular systole and proisystole are coupled (bigeminism), we might conclude, if we had Leads I and III only, that the phenomena were the same. Thanks, however, to Lead II which enables us to identify the P wave, we see that, while the ventricular series preserves the same appearance in both curves, the auricular series differs in the two; in figure 74 the P rhythm is regular, but in figure 73 it is bigeminal, the P waves being doubled. The independence of the two series is thus manifest.



FIG. 75. Allorhythmia due to ventricular protosystoles. The systoles and protosystoles, R_1 , combine as couplets, three of which are seen. The auricular series is regular.



FIG. 76. Allorhythmia due to ventricular priosystoles. The allorhythmia is here more complete than in the preceding figure. The systolic-extrasystolic couplets occur in threes, forming two composite groupings of the same physiognomy. The regularity of the auricular series is in marked contrast to the irregularity of the ventricular.

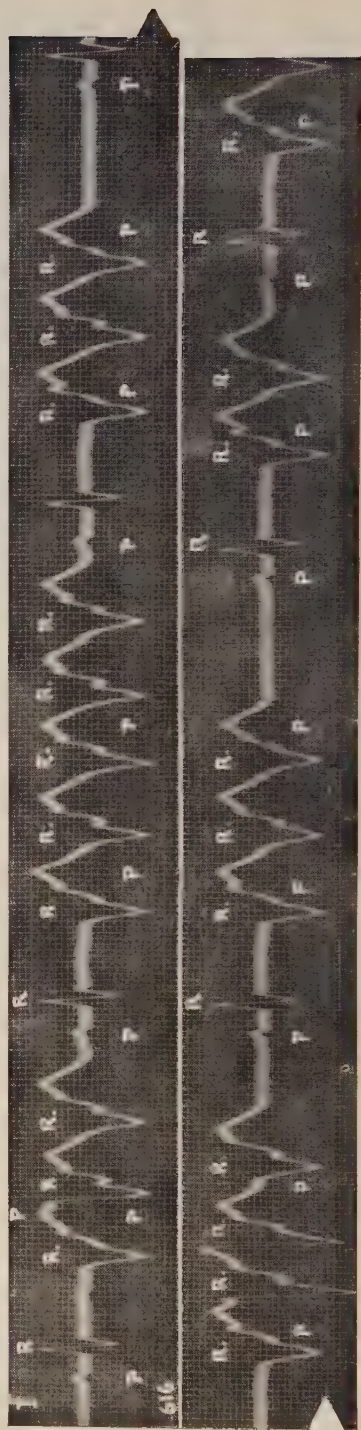


FIG. 77

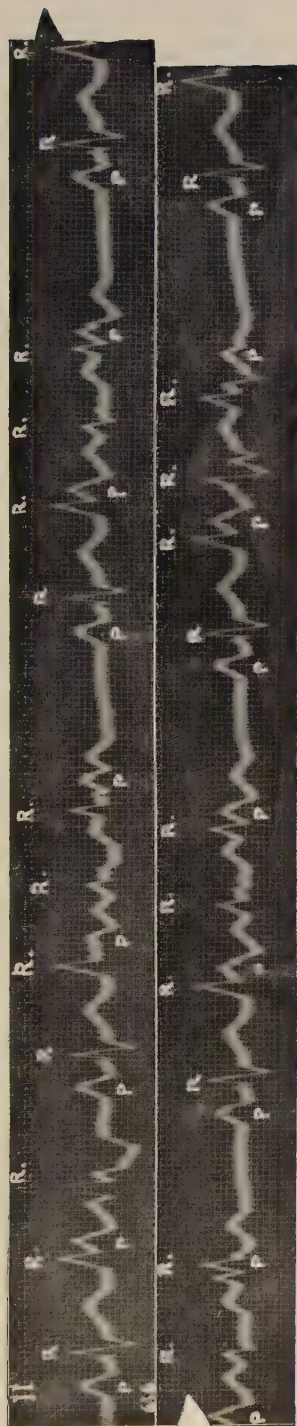


FIG. 78

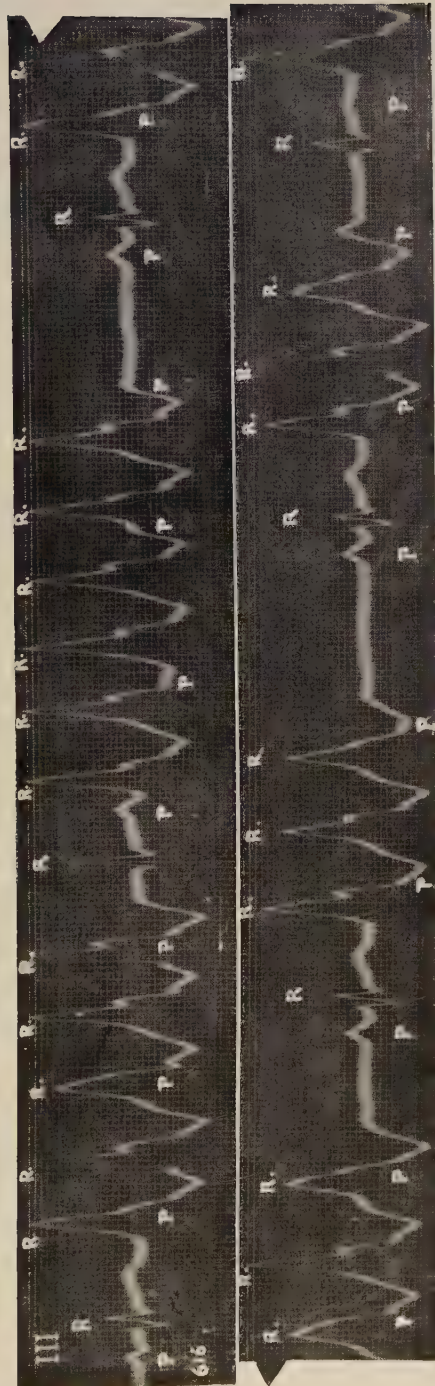


FIG. 79

FIGS. 77, 78, AND 79. Volleys of ventricular proisystoles of the same type. The identification of the auricular series can be made only in Lead II. In Leads I and III they are doubtful. These tracings are from the same patient as those in figures 73, 74, and 76.

Ventricular Proiosystoles

The ventricular series is broken by itself much more frequently than is the auricular series, the latter at the same time remaining regular. It is necessary to make an attentive study of the tracing to reconstruct the regular series of P waves, some of which are submerged in the ventricular complexes. We are sometimes able to identify the P waves by noting first the auricular rhythm. It is correct always to recall that this rhythm is never mathematically regular and the interval PP may vary by some hundredths of a second.

Comparison of the tracings taken in the three leads is often very useful. One of the leads may reveal the event P clearly, while its existence remains doubtful in the other leads (figs. 73 and 74). Theoretically one should compare two leads registered simultaneously with a two-string galvanometer. But in practice the proiosystoles are very often repeated with regularity so that with the preceding systoles of the same series, they constitute a typical grouping which is found as identical in the two leads taken at a few seconds interval. Consequently when in one lead P appears in a group in the middle of the ventricular complex, it is legitimate to conclude that in an identical group in another lead, P really exists in the same relative position. It does not appear there because in this lead the events of the ventricular complex have a different morphology and thereby mask it.

Ventricular proiosystoles with normal or deformed complex may appear under various aspects. The most simple case is that of a single proiosystole breaking the auricular series once. The systole which precedes it with the proiosystole itself form a sort of couplet that contrasts strikingly with the rest of the curve. This couplet may appear at irregular or regular intervals and between the couplets there are intercalated a variable number of normally spaced systoles. If the couplet reappears at regular intervals the curve assumes a sort of rhythm. This regularity in the irregularity of the rhythm is sometimes characterized by the term *allorhythmia* (figs. 75, 76).

When the systole-proiosystole couplets succeed each other regularly without the interposition of uncoupled systoles, we have *bigeminism* (fig. 74); this is frequent in *digitalis* poisoning.

The proiosystole of each couplet may have the same deformity or it may be deformed according to two types which alternate regularly (fig. 134).

Many proiosystoles may follow one another, forming with the normal systole, which precedes the first of them, a more complex group. Thus two proiosystoles form, with the antecedent normal systole, a ternary group, and we have *trigeminism*.

The proiosystoles may be deformed in the same or in different ways.

Figures 77, 78 and 79 show a series of ventricular proiosystoles in which the deformity of the complex is always the same. Figure 80 shows successive proiosystoles the deformity of which differs. In each grouping the different forms always dispose themselves in the same order.

The proiosystoles may lose all regularity. There is no longer allo-rhythmia but arrhythmia. The ventricular series is broken at each moment and in varying fashion. In these cases the complexes often differ in form.

Conjoined Auricular and Ventricular Proiosystoles

A rupture of both the auricular and the ventricular series by proiosystoles is often encountered, so that there is a combined auricular and ventricular proiosystolia (figs. 81, 82 and 83). The complexes are not habitually deformed although they may be.

The initial phase of the P complex may keep its ordinary direction, but its amplitude and form change. Figure 71 (note the fourth P) gives an idea of this alteration. Or the initial phase of P may be changed in direction, whence the aspect known as negative or inverted P (figs. 81, 82, and 83).

In the conjoined proiosystole P and R may be unequally premature, their reciprocal relations varying. Most frequently P continues to precede R, whence the combination qualified classically as "auricular extrasystole." It is the type shown in figures 81, 82, and 83. At times P is premature, markedly displaced upstream, and is no longer distinct but is swallowed up in the T of the preceding complex. It appears distinctly only in Lead III (fig. 84). Finally it may happen (fig. 85) that P being relatively less premature than R, remains beyond R, whence the combination which should be called "infranodal atrioventricular extrasystole." But this would be an "infranodal extrasystole" of exceptional type, for the ventricular complex is deformed in it, when, according to the admitted hypothesis, it should be normal; for the stimulus of the extrasystole is supposed to arise upstream from the bifurcation of the bundle of His (fig. 85).

We find in the conjoined rupture of the two series, auricular and ventricular, the same varieties as those enumerated when there was a rupture of a single ventricular series, namely, the isolated type (fig. 81), the allorhythmic type with its bigeminal and trigeminal varieties (figs. 82 and 83), and the arrhythmic type properly speaking.*

It is always the same mechanism which enters into play, but just before

* In the charts the author distinguishes between the systolic and the proiosystolic waves P, R, and T, by marking the latter P., R., and T.; for greater distinctness the translator has changed these proiosystolic signs, in the text, to P', R', and T'.



FIG. 80. Ventricular preexcitation with a delta wave and a premature R wave (R') preceding the main R wave. The tracing is labeled 'III' and '572'.

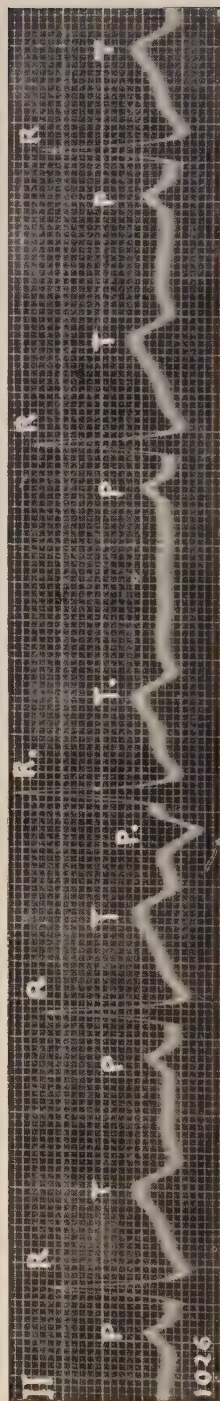


FIG. 81

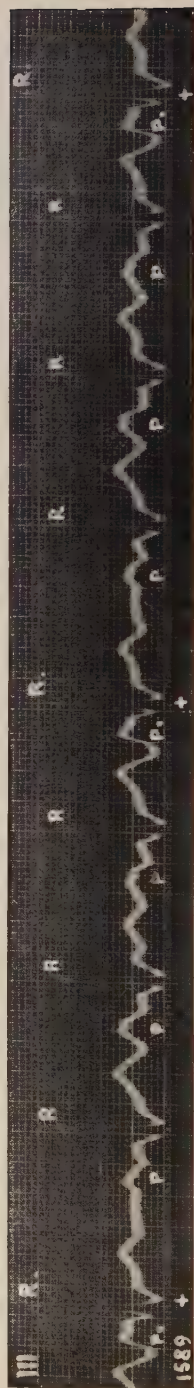


FIG. 82

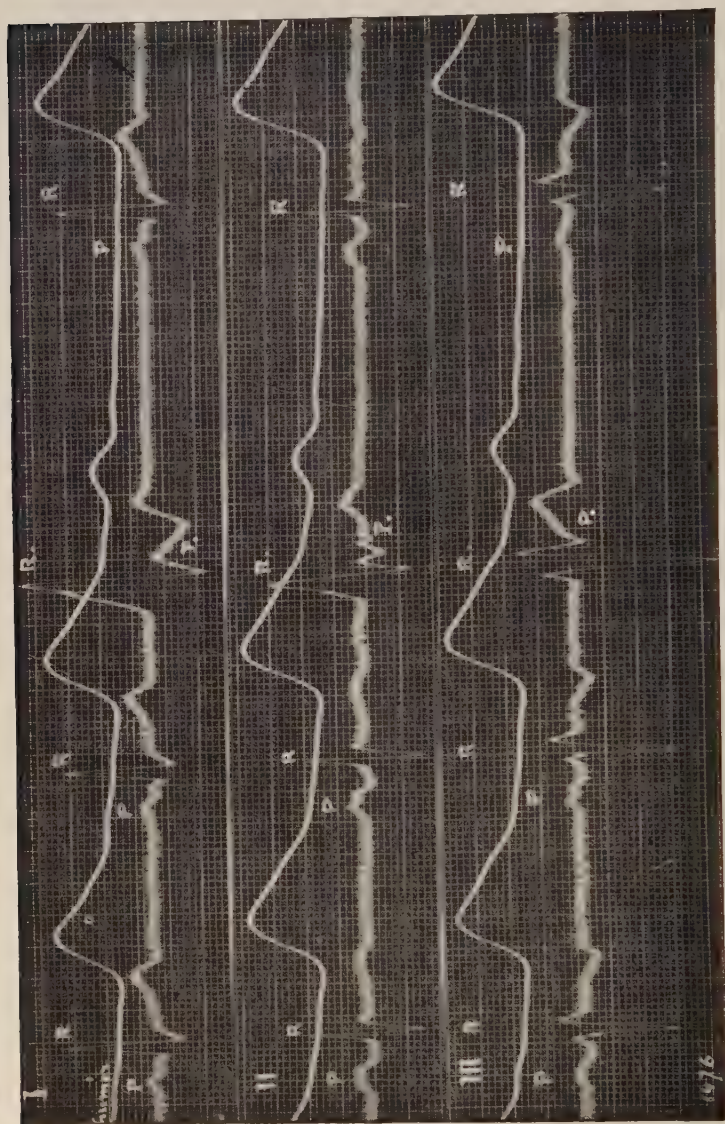


FIG. 85. Conjugate auricular and ventricular preexcited beats. The auricular series, as well as the ventricular, is interrupted by one preexcited beat; but, the ventricular preexcited beat being earlier, the grouping formed is R.P., and not P.R., as in the preceding cases. A very diminished brachial wave corresponds to the ventricular preexcited beat, too near which is the preceding ventricular systole.

the ventriculonector was alone involved, while now the atrionector and ventriculonector are jointly implicated.

We have just recounted the facts discovered in reading separately the auricular and ventricular series; each of the premature systoles has been considered in relation to the systoles of the same series. It is as if we had divided the electrocardiogram, placing on an upper line the auricular complexes and on the lower the ventricular complexes.

But as we have seen, it is not thus that an electrocardiogram is classically deciphered. Auricular and ventricular complexes are considered as belonging on the same line. The result is that the idea is formed of a relationship between the auricular and adjacent ventricular complexes. It is at this point of view, at the unilinear point of view one might say, that one stands when speaking of extrasystoles.

I shall give later a series of tracings which go to show that this unilinear grouping is wholly arbitrary. In fact a slight variation of one of the two series, auricular or ventricular, or both, suffices to modify the apparent grouping between a P and an adjacent R. The independence of the two series appears then in all its clearness.

The curves which follow, when studied with the preceding, may be assembled in two groups. In the first the independence of the two series, auricular and ventricular, is rendered manifest, in favor of a variation of the rhythm of the ventricle, by ventricular proiosystolia; these are the tracings 78, 79, 80, 86, 87, 88, 89 and 90. The ventricular series is broken by proiosystoles which are more or less premature, followed by longer or shorter post-proiosystolic pauses. It results therefrom that the events P and R (systoles) and R' (proiosystoles), if one persists in trying to establish a connection between them, assume the most varied relations. The particular P will be coupled with R', following different modes which may be called auricular, atrioventricular, ventricular, interpolated systoles, etc. Such a classification seems to us as artificial as useless.

The curves 88 and 89 (taken in the same subject at a few seconds interval) showing the time taken in passing from L. II to L. III, are interesting. The auricular series is regular in both tracings; the rhythm of 114 in figure 88 is slightly reduced to 100 in figure 89. The ventricular series in figure 88 is broken twice by a proiosystole without any allorhythmic type being in evidence. The impression is given that P and R are always coupled, the interval PR' simply being shorter than the PR interval. Suddenly the ventricular rhythm changes and there is allorhythmia of the bigeminism type while the ventricle is slowed. The discordance between the ventricular series, so changed, and the auricular series, so little modified, is manifest. There is no longer any possible grouping between P and R or P and R'.



FIG. 86



FIG. 87

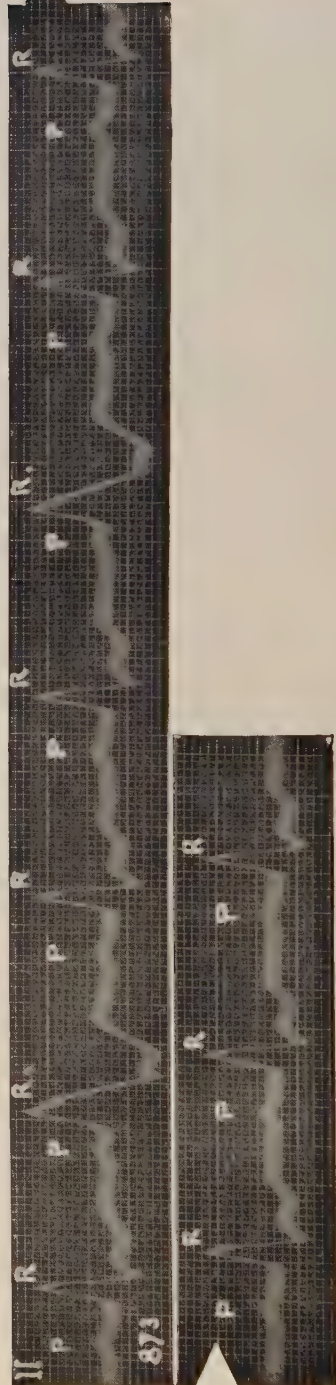


FIG. 88

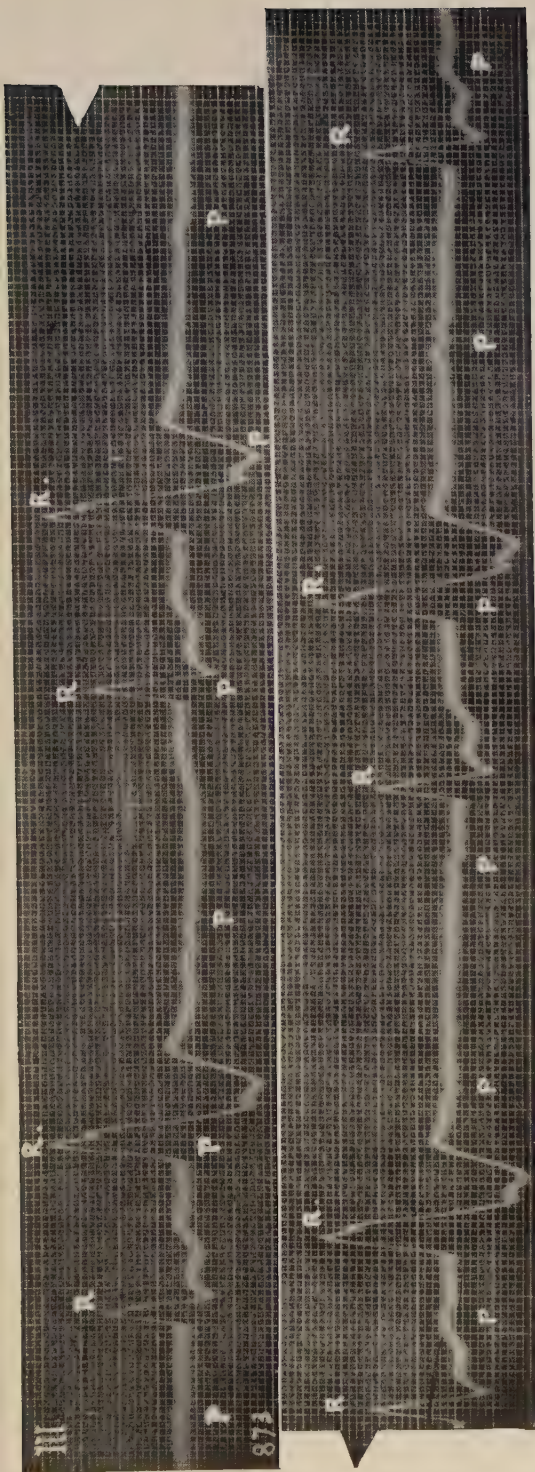


FIG. 89

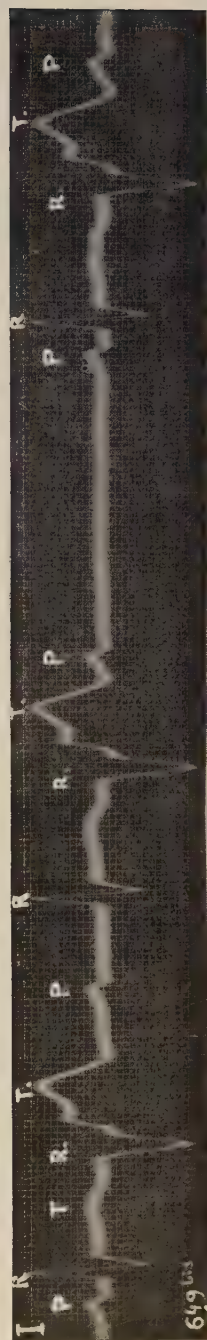


FIG. 90

FIGS. 86 to 90. Independence of the auricular and ventricular series in the course of an attack of ventricular proisystoles. The auricular series is regular, the ventricular one is broken by the occurrence of more or less premature proisystoles followed by longer or shorter pauses. If one nevertheless wishes to connect the P and R waves one will obtain the most varied combinations certain of which correspond to some of the varieties of the classical "extrasystoles." (Figure 86 is from a patient who has already given the tracings shown in figures 73 to 79.)



FIG. 91

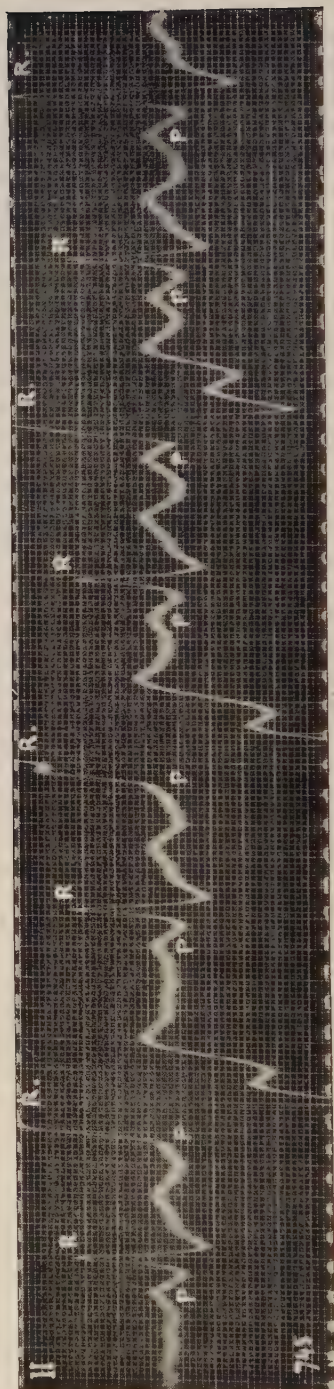


FIG. 92

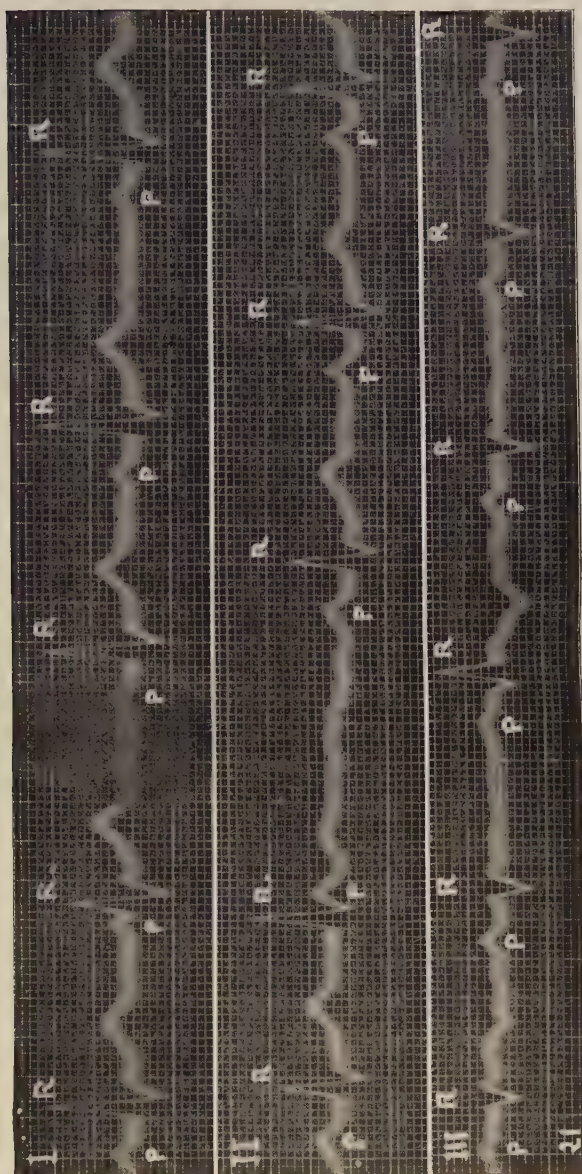


Fig. 93

In the second group we have assembled the curves in which the independence of the two series, auricular and ventricular, appears under the aspect of a simultaneous variation of the auricular and ventricular rhythms, produced by auricular and ventricular proiosystoles. These are the tracings in figures 91, 92 and 93. It will be remarked on the tracing in 91 that the first proiosystole R' comes later, relatively to the systole R which precedes it, than does the proiosystole R' of the second half of the tracing. Further,

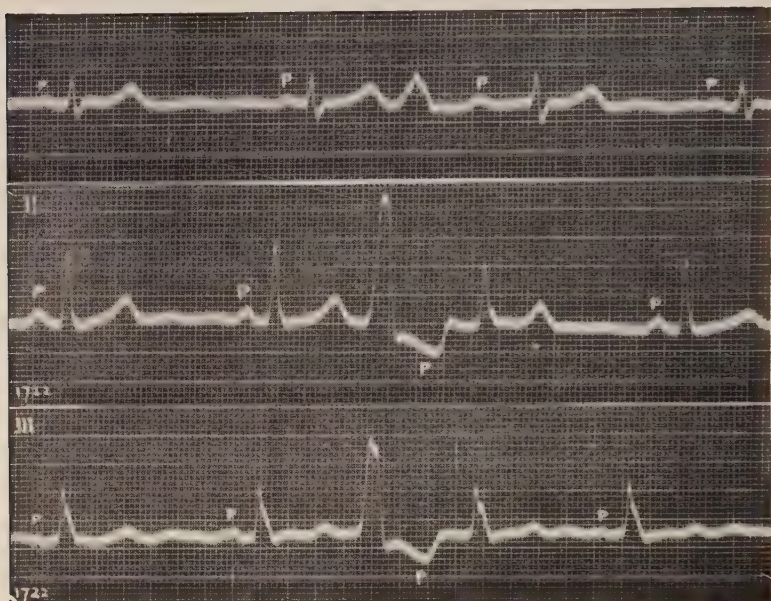


FIG. 94

FIGS. 91 to 94. Independence of the auricular and ventricular series in the course of an attack of conjugate auricular and ventricular proiosystolia. Both series are broken by proiosystoles, but the latter are more or less dissociated "up-stream," from which it follows that the P. and the R. waves have various relations. In figure 94 the third R corresponds to the type called "interpolated extrasystole" (see the text).

the proiosystole P' is not yet engaged in the first R' while it is so in the second. A slight dissociation between the auricular and ventricular proiosystoles of the two groupings has sufficed to produce this. Classically one would deduce therefrom that there are two extrasystoles of different type, the more so because the aspect of the ventricular complex differs equally in the two groupings. The same may be said of tracings 92 and 93. Tracing 94 shows that the auricular series is broken by a proiosystole P'

(the third P). The ventricular series is broken by two proiosystoles, the first alone having a complex of anomalous form. It is of interest to compare this episode with that which is a prelude to the crisis of auricular tachy-rhythmia in figure 70. Here are the same proiosystolic deformity, the same supernumerary proiosystole, but in figure 94, the proiosystolia is ventricular and in figure 70 the supernumerary proiosystolia is auricular. If one interpret the episode in figure 94 as an abortive crisis of tachyrhythmia, one will note that the tendency to ventricular tachyrhythmia is more evident in L. III than in L. I and L. II.

The Pulse in Proiosystolia

If we take the arterial pulse at the same time as the electrocardiogram, we will note that the ventricular proiosystole corresponds to a diminished beat which may be so attenuated that it cannot be felt at the wrist, or if the radial pulse can be felt under the finger, it no longer registers exactly the ventricular beats (fig. 85). But we note, on the other hand, that this is not the case in figure 95, for all depends on the interval which separates the systole from the proiosystole, as compared with the preceding intersystolic interval. If the first of these intervals is relatively but little shortened, the pulse is not affected, while if it is greatly shortened there is considerable weakening of the pulse beat.

THE HYSTEROSYSTOLES

To the proiosystole or premature systole is opposed the hysterostyle or retarded systole which results from a diminished activity of the cardionector. This modification seems to us to be related almost exclusively to a variation in the circulation of the cardionector. But while in the proiosystole the circulatory régime is probably modified, on either the arterial or the venous side, in the hysterostyle only the arterial side is involved. A diminution of the arterial delivery seems to us to be the cause of the hypoactivity of the cardionector, whence the slowing of its rhythmic function. We are going to analyze an entire series of tracings showing hysterostyles.

The first tracing is an example of ventricular hysterostolia in the course of a ventricular bradyrhythmia (fig. 96) combined with an auricular tachyrhythmia, the relation being $\frac{450}{20} = 16.07$. The interval between the systoles of the ventricle is habitually 3 seconds. The tracing shows the rupture of the ventricular series by a hysterostyle separated from the preceding systole by a pause of 6 seconds. We believe that the ventricular bradyrhythmia is related to a stenosis of the artery of the ventriculo-

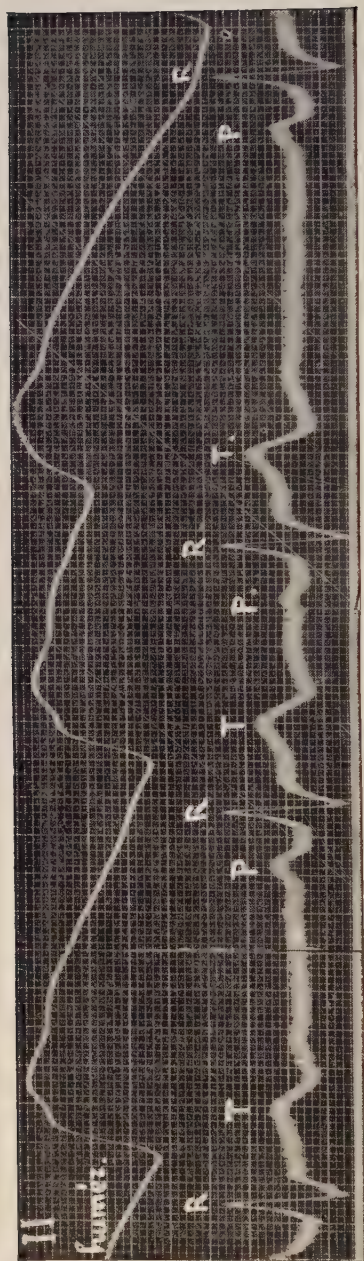


FIG. 95. The arterial pulse in proiosystole. The brachial curve does not fall after the ventricular proiosystole because the slowness of the rhythm has given time for the diastolic filling of the artery between the normal systole (the second R) and the proiosystole R. There is here a simultaneous proiosystole of the auricle, P., and the ventricle, R. In figure 85, which should be compared with this, the brachial curve shows the influence of the premature contraction of the ventricle by a very slight wave only, for the reason that the rate is more rapid and therefore the time clapsing between the systole and the proiosystole is not long enough to permit the filling of the ventricle to the point where its discharge will appreciably distend the vessels.

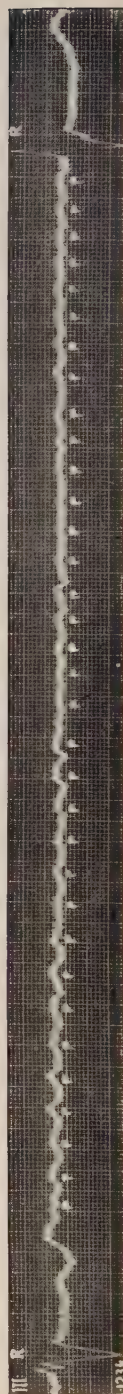


FIG. 96. Ventricular hysterosystole of 10 seconds' duration in a patient with ventricular bradyrhythmia and auricular tachyrrhythmia

nector and the hysterosystole to a momentary augmentation of the stenosis by a tonic contraction of the muscular tunic of the artery. The auricular tachyrhythmia is probably imputable, as we shall see later, to a venous stasis in the atrionector.

When, instead of remaining constant, the ventricular hysterosystolia increases progressively, beginning with a group PR taken as origin (for the dissociation of P and R remains at average values), the dissociation increases regularly until at a certain instant there appears to be a rupture in the groupings of P and R; R seems as if isolated and then a P supervenes, likewise isolated. Then we note again the grouping PR which inaugurates a new period. It is this isolated P which the English term a "dropped beat," that is a beat which drops as if there had been one in excess. The phenomenon may result, not from a progressive hysterosystolia but from a sudden hysterosystole following a proiosystole.

When there is a double hysterosystole, but more marked in the auricle, the ventricular hysterosystole appears before the auricular and it seems as if the ventricle were beating on its own account, that there was, so to speak, a "ventricular escape."

COMBINED PROIOSYSTOLES AND HYSTEROSYSTOLES

Proiosystoles may combine, either among themselves or with the systoles, and the two series, auricular and ventricular, may be broken in various ways. The suppositious groupings of P and R then take on all aspects, and all combinations are possible. Figure 93 shows one of these. Besides the ventricular proiosystoles, unequally premature, in L. I and L. II there is in L. II an auricular hysterosystole.

The curve in figure 97 offers a new example of these combinations. The upper tracing shows auricular and ventricular hysterosystolia coming on abruptly and breaking from time to time the regular series of the auricular and ventricular complexes beating at a rate of 72. Suddenly there is a suspension of both auricular and ventricular pulsations, and after a pause of 2.6 seconds the rate of 72 is resumed for both series with the same dissociation PR. All happens as if the cardiac mechanism had been wholly and abruptly arrested. We have seen that this phenomenon was called sinusal block. It is assumed that the pneumogastric inhibits the sino-auricular node. But if we study the lower tracing in the same figure taken in L. III on the same patient and a few seconds after the upper tracing, we find that the aspect is very different. The ventricular series is broken because of the ventricular proiosystoles and hysterosystoles; at one moment there is an abortive renewal of the rhythm at 80, that is at something near



FIG. 97. Combined auricular and ventricular protosystoles and hysterosystoles. The upper line gives the impression of a close relation between the auricular and ventricular rhythms; the lower tracing, taken some seconds after the upper, reveals their independence.

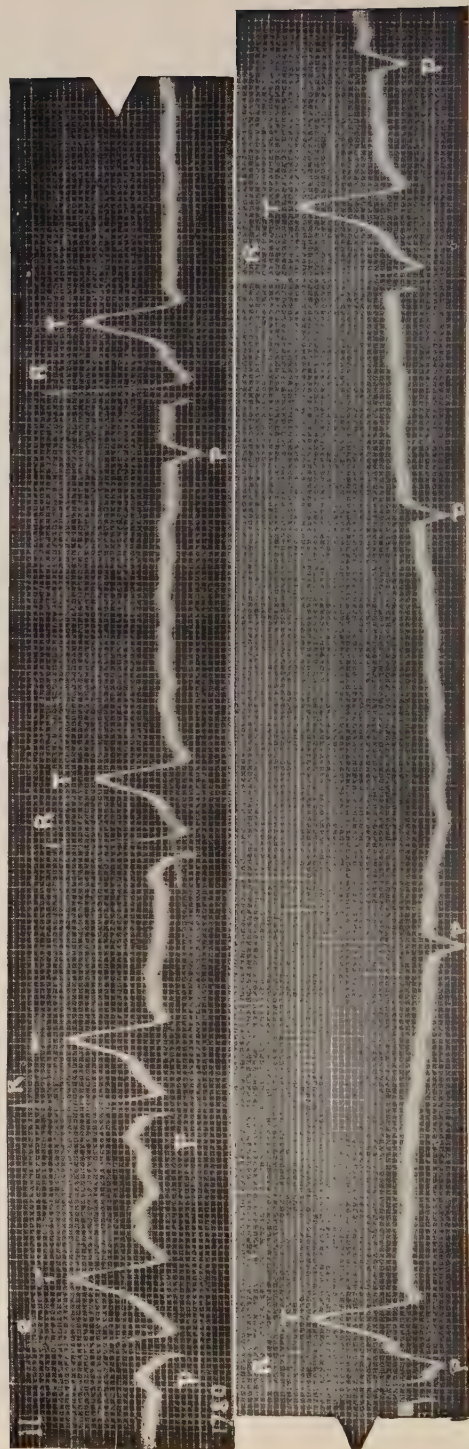


FIG. 98. Disassociated effect of ocular compression on the two rhythms. The auricular and ventricular rates are both modified, but the ventricular hysterosystole is much more marked than the auricular, their independence being therefore evident.

the normal rate, well defined in the upper tracing. The auricular series is also irregular and follows a disposition analogous to that of the ventricular series. But—and this is an important fact—the appearance of a grouping between the neighboring auricular and ventricular complexes has vanished. If then the upper tracing could be strictly interpreted as indicating a momentary action exerted by intermediation of the sinus node on the blocked heart, the lower tracing can be considered only as translating two different actions, one on the auricle and the other on the ventricle. This dualism is thus unmasked on one of the tracings and there is a great likelihood that it is latent on the other. The same cause acting on the two cardioneotors, given that its action is sometimes the same, sometimes different, explains the variation of the phenomenon. If the cause acts by intermediation of the circulation, which we suppose to be often the case, a vasomotor change bearing equally on the entire arterial system would correspond to the first tracing, and one unequally distributed among the arteries would correspond to the second. The passage from one to the other, which a film taken at a favorable moment might show, squares well with a circulatory variation.

Compression of the eyeball, as all know, slows the action of the heart. It is not a matter of action on the whole heart but on the auricle on the one hand and the ventricle on the other. The hypothesis of a sinus block cannot be sustained. Figure 98 is particularly illustrative, for the auricular and ventricular rhythms have both been slowed, and slowed unequally (see also fig. 129).

ALTERNATING PHASES OF PROIOSYSTOLES AND HYSTEROSYSTOLES

The auricular and ventricular series may be jointly modified in such manner that during a first phase the systoles succeed one another rapidly, in a second phase more slowly, and so on. This alternation in the method of functioning of the auricles and ventricles seems in certain cases to be connected with alternation of the respiratory phases, and it has been said that a respiratory arrhythmia is present. I give here three examples of this. The stage of acceleration accompanies the entire expiratory phase on the tracing in figure 99, and all of the inspiratory phase in the tracings in figures 100 and 101, scarcely running over into the expiratory phase. The rate passes respectively from 37 to 76, 66 to 120, and 54 to 90.

An attentive study of the tracings shows on the other hand that at the moment of acceleration, the auricle beats more rapidly than the ventricle. Conversely it beats more slowly than the latter at the time of slowing. The result is that the interval PR lengthens when the rhythm accelerates and



FIG. 99. Alternate phases of auricular and ventricular proisystole and hysterosystole ("respiratory arrhythmia")

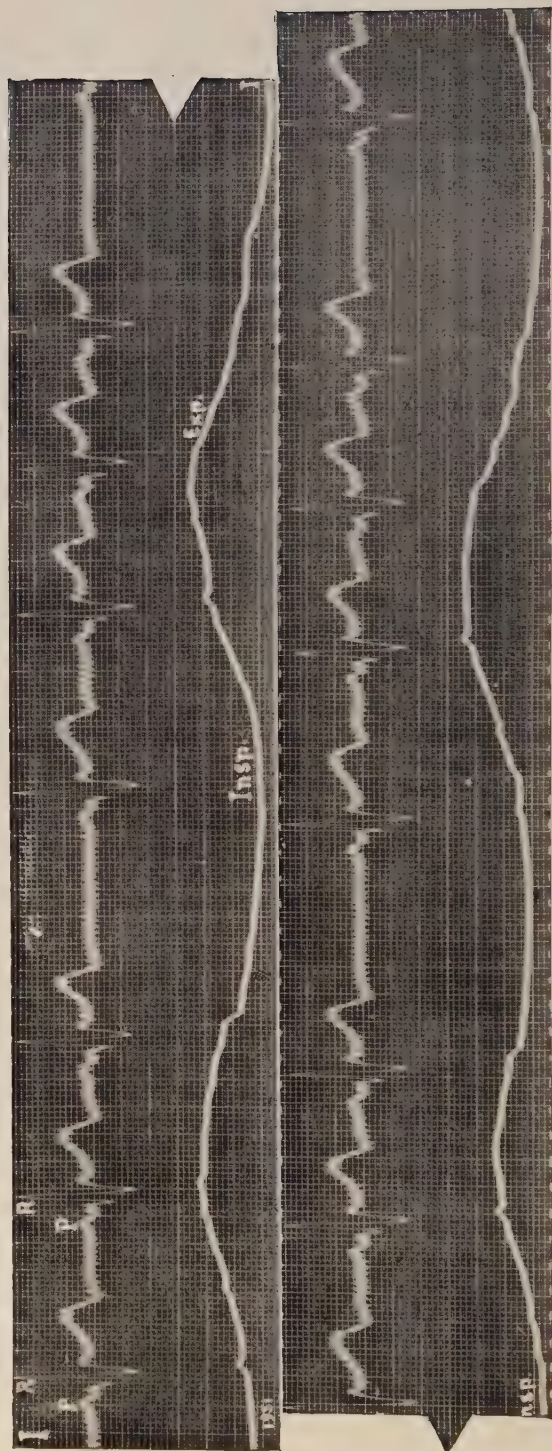


FIG. 100. Alternate phases of auricular and ventricular proisystole and hysterosystole ("respiratory arrhythmia").

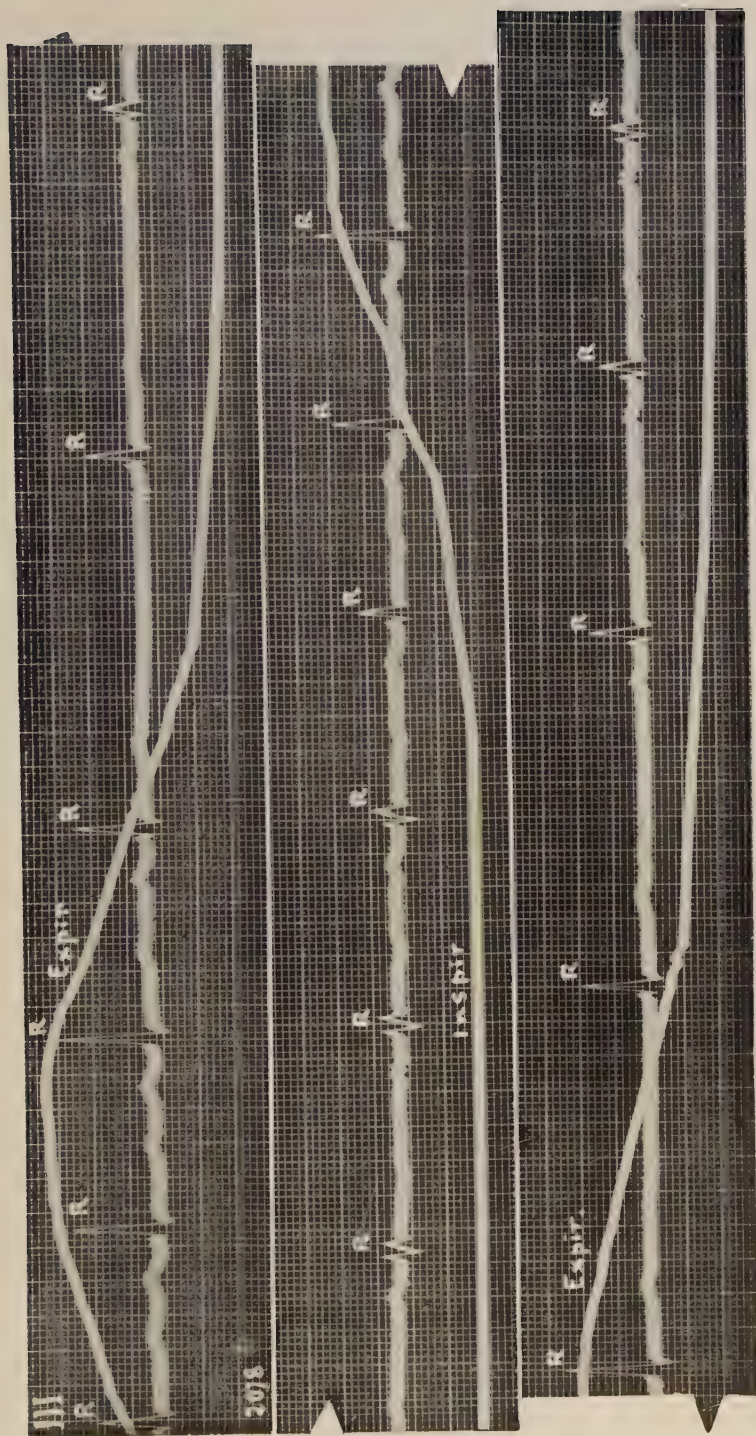


FIG. 101. Alternate phases of auricular and ventricular proisystole and hysterosystole ("respiratory arrhythmia").



FIG. 102. Alternate phases of auricular and ventricular protosystole and hysterosystole. In this tracing we see successively the end of a crisis of auricular and ventricular tachyarrhythmia, the return to the normal rate, and the beginning of a new tachyarrhythmic crisis. It will be seen that in the period of slowing down, the auricle retards more than the ventricle, whence the sudden narrowing of the PR interval; when the rapid action begins again the auricular beat is accelerated more than the ventricular, whence the sudden widening of the P.R. interval. The auricle appears each time to react more to the perturbing cause of the rhythm. RP, RR, duration of action of the atrionector and the ventriculonector respectively.

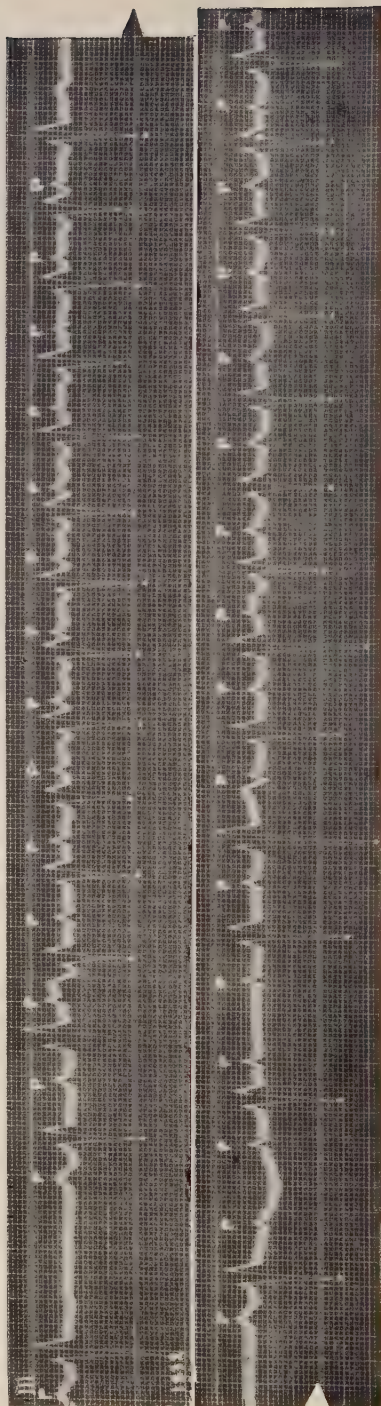


FIG. 103. Alternating phases of auricular and ventricular protosystole and normal rhythm

diminishes when it slows, a phenomenon the reverse of that seen in simple "bradycardia" and "tachycardia," previously studied, where PR varied inversely with the rhythm.

It is then doubly inexact to say that in respiratory arrhythmia there is an expiratory bradycardia. There is no bradycardia, for it is not the entire heart which is slowed but the auricle on one hand and the ventricle on the other. There is a coupled bradyrhythmia, the slowing of auricle and ventricle being unequal.

The bradyrhythmia is not expiratory and the alternation of the phases of acceleration and retardation is sensibly parallel with the alternation of the respiratory phases. But there is not a concordance such as when the expiratory phase corresponds always with the phase of slowing.

We have to do here undoubtedly with a very complex phenomenon. We see in it the expression of a variation of the circulation in the cardioneotors, a variation which is probably chiefly caused by an alteration of the pressure in the right auricle as a result of the respiratory movements. From this results a hyperemia by stasis in the cardioneotors and acceleration of the corresponding rhythms. This is the case especially in the tracing shown in figure 100.

But in other cases the variation in the amount of blood is due especially to a diminished delivery of the artery. The tracings in figures 99 and 101 square better with this hypothesis, for the series of complexes is broken by retardation, and no longer by acceleration as in the preceding tracing.

The alternating phases of acceleration and retardation may bear no relation to the phases of respiration. The rates are altered abruptly, becoming either more rapid or slower, during a longer or shorter interval. The phenomenon strongly resembles the preceding and is probably due to an analogous mechanism—venous stasis of auricular origin if there be acceleration, ischemia of arterial origin if retardation.

Paroxysmal acceleration of the heart beat enters into the great class of "tachycardias." The tracings which we give serve after a fashion to illustrate the passing between the proiosystoles and the tachyrhythmias. The tracing in figure 102 corresponds to a complete paroxysm of rhythmic acceleration. At its beginning we note an elongation of the PR interval and there is also a shortening of PR when the rate is slowed. Here again these variations in rhythm maintain, therefore, their independence. This cannot have to do with an action bearing on the sino-auricular node alone, for the two cardioneotors are involved, each on its own account. The respiratory phases registered on this tracing have no relation to the cardiac phases. The tracing in figure 103 is an example of a paroxysm of increased

rapidity of the heart beat. A complete paroxysm and the beginning of another have been registered. It will be noted that each access begins in the same way. A proiosystole is coupled with a systole in each of the series; the proiosystole of the ventricle is here in a deformed complex. This couple appears twice in the lower half of the tracing. Furthermore the independence of the two rhythms is manifest. This is shown first by the variation of PR, and again where, during the second paroxysm, the auricular tachyrhythmia occurs before the ventricular.

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CHAPTER VII

ANOMALIES OF THE CARDIAC MECHANISM DUE TO MODIFICATION OF THE PR INTERVAL

When we give the electrocardiogram a bilinear reading, the PR interval is of importance only in the mathematical sense; it measures the dissociation between the auricular and the ventricular systoles. But according to the classical theory, when the reading of the electrocardiogram is unilinear, the PR interval represents the time which the sino-auricular stimulus takes to excite the ventricular systole. From this hypothesis conclusions have been deduced, which we shall discuss, touching the rhythm of the heart as a whole.

In the normal heart P precedes R and the PR interval measures on an average 10/100 to 15/100 second. In anomalous cases it may assume any value and the values may be positive or negative; in the latter case the sequence is RP, the ventricle beating before the auricle.

We shall study, therefore: (1) The cases in which PR has a value lower than the average, comprising the cases in which this value is nul or becomes negative; (2) Cases in which PR is above the normal in value.

WHEN THE INTERVAL PR IS LESS THAN THE AVERAGE

If PR represents the time which the stimulus, leaving the sino-auricular node, takes to arrive at the auricle, then when PR is below the average it may be assumed that the stimulus travels a shorter course, and that as a consequence it must have originated below P. For the sino-auricular center, the major center, there has now been substituted some one of the auxiliary nervous centers seated below it.

It is on the basis of the same fact that it is assumed, according to the classical theory, that the heart functions as a whole, with all its parts closely connected; thus the idea of a heart rhythm is always considered legitimate.

When PR has a mean value, 10/100 to 15/100 second, the rhythm is called sinus; this is the normal cardiac rhythm, dominated hypothetically by the major sino-auricular center.

We say that there is a juxta-sinus or auricular rhythm when the PR interval is notably diminished, without however specifying the point below which one may speak of diminution. It is admitted that when the stimulus originates in an auxiliary center P is sometimes negative.

It is said that there is an atrioventricular, nodal, or septal rhythm when P is very near R, either in front of or behind it, or is consolidated with R. This disposition of P and R is considered as proof that the stimulus originates in the node of Tawara, passing upstream in retrograde direction to cause the contraction of the auricle, and also downstream to excite contraction of the ventricle. If PR remain positive it is said that the stimulus originates in the higher part of the node of Tawara; there is a supranodal rhythm. If PR is suppressed, P coinciding with R, it is because the stimulus originates in the middle portion of the node; the rhythm is nodal, properly speaking. When PR is negative the stimulus originates in the lower portion of the node; the rhythm is infranodal.

In all of the varieties which we have just enumerated the rhythm is controlled by a minor auxiliary center. The major center is assumed to be inefficient or inactive.

WHEN THE INTERVAL PR IS GREATER THAN THE AVERAGE

The elongation of PR is explained by an obstacle in the conduction of the stimulus leaving the major sino-auricular center and reaching the ventricle only after a certain delay. We cannot determine precisely the figure, departing from which the sequence PR becomes abnormally elongated. It is agreed that if the interval exceeds 20/100 second the elongation becomes abnormal and this is all that can be said.

When the interval PR lengthens considerably, the impression is given that there is a rupture between P and R and that the ventricle contracts on its own account. It is said that the stimulus leaving the auricle is arrested, like a train before the signal light of the block system. There is a block.

A second stimulus is thought to be furnished by a minor auxiliary center. This center, situated in the bundle of His above the bifurcation, affects the ventricle only and the rhythm is called idioventricular, but it differs from the preceding cases in which there was only one cardiac rhythm, for here there is no longer a single cardiac beat but two beats, one for the auricle and one for the ventricle. The former is the sinusal rhythm, no longer commanding the entire heart, but limited to the auricle. The rhythm for the ventricle is a superadded one. We shall encounter these facts again when we study the anomalies of the cardiac mechanism characterized by discordance of auricular and ventricular rhythms.

SIGNIFICANCE OF THE PR INTERVAL

The meaning of PR seems to us to be very different from that of the conduction time between auricle and ventricle. The dissociation of PR would

appear to indicate the difference which exists between the beginning of the systole of the auricle and of that of the ventricle. This difference depends on the disposition of the arterial system supplying the atrionector and the ventriculonector. Whatever may be the variety of origin of the arteries of the cardionectors the course of the artery from the aorta to the atrionector is shorter than the corresponding course to the ventriculonector. Moreover, all things being equal, the wave of blood propelled by the left ventricle will make its effect felt sooner on the atrionector than on the ventriculonector.

We would not say that it is the wave which elicits the auricular and ventricular contraction, but we do say that this wave, by the sudden increase of the blood supply in the cardionector, effects the rhythmic activity of the latter—one might say the fact of its rhythmicity. It makes the circulatory flood-tide which is necessary for the initiation of the action of the cardionector, and makes it earlier in the atrionector than in the ventriculonector. We shall see later that if any obstacle diminishes the output of an artery, the rhythmicity of the corresponding cardionector will be diminished and that there will be a retardation of its getting into action.

We do not imagine, however, that the ventricular wave determines the contraction which follows its arrival. Nevertheless we believe that it has an influence at the moment of its production, as we shall indicate when we come to study the so called Stokes-Adams syndrome.

The sudden increase of the blood supply depends probably on the propulsion of the blood wave at the moment of the ventricular systole, but it depends also on another factor—the contraction of the artery of the cardionector. A variation of the activity of the muscular tunic of that artery, should, it seems to us, be considered. This modifies the local circulation more or less and to that extent the action of the cardionector. We have emphasized this action of the arterial musculature upon the blood supply in the work done by us in collaboration with Dr. A. Luisada, mentioned above.

These reservations made, if the blood flow is equal in amount in the atrionector and ventriculonector, the auricular and ventricular beats will be equal, the interval between them will remain constant, and PR will be the measure of this interval.

If there is a disturbance of the circulatory equilibrium a precocious action will correspond to an augmented discharge and a retardation to a lessened discharge. The systole of the cardiac cavity under the control of the nector will be either advanced or retarded and there will be a proiosystole or hysteriosystole.

The PR interval, normal when it marks the time between the auricular

FIG. 104

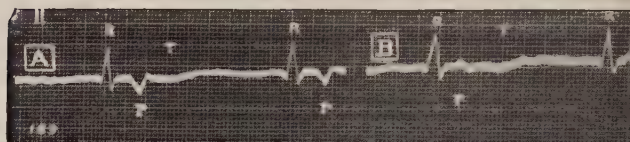
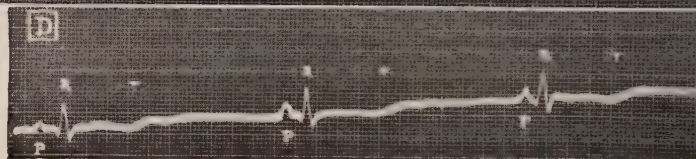


FIG. 105



FIG. 106



FIGS. 104, 105, AND 106. Experimental variation in the chronology of amyl nitrite. In these three figures I have faithfully reproduced the Donzelot; but I have grouped the fragments of the film differently, taking view of the entire phenomenon. The fragments A, B, C, D, E, are in re

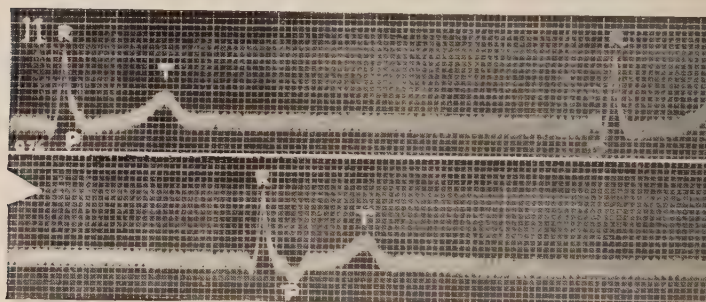
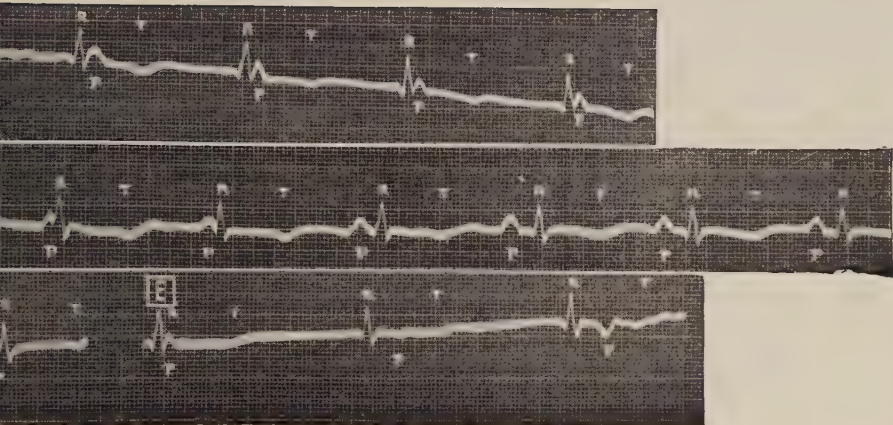


FIG. 107. Spontaneous variation of



ations of P and R by unequal acceleration of the rhythms under the action
 ggs previously published in the article written in collaboration with Dr.
 unt of the undulations of the curve in such a way as to give a more striking
 to some of the principal successive phases.



ronological relation of P and R.

and ventricular systoles, will become abnormal when it corresponds to the time separating a systole from a proiosystole or from a hysterosystole.

In order to clarify our ideas let us take, to start with, an event R or R_0 . The rhythmicities being normal, we first see an auricular systole P_1 supervene, then after a given interval, P_1R_1 , a ventricular systole, R_1 . R_0R_1 measures the time of the ventriculonector. This designation does not signify that R_0R_1 is the time required for the ventriculonector to get into action, for we do not know the length of this time; it signifies rather that the getting into action has supervened after this time, R_0R , reckoned conventionally as starting from R_0 .

R_0P_1 will measure the time of the atrionector, under the same conditions.

PR is the difference between R_0R_1 and R_0P_1 . Normally this difference is positive. It has an average value, for it may vary in different subjects according to the anatomical conditions of the coronary system, in particular according to the differences in origin and course of the arteries of the cardionectors. This difference will vary with the same subject if in place of being the difference between two systoles it represents the difference between a systole and a premature or retarded systole.

Suppose an obstacle diminishes the discharge of the artery of the atrionector, the time R_0P_1 will lengthen. On the other hand R_0R_1 does not change and the difference P_1R_1 will be less. This is the dissociation between an auricular hysterosystole and a ventricular systole. Because the obstacle increases for the artery of the atrionector, P_1R_1 will diminish still more, may be extinguished, or even become negative.

We may reason in the same manner by assuming that it is the discharge of the artery of the ventriculonector which is diminished. In this case, it is PR which is lengthened, for this is the dissociation between an auricular systole and a ventricular hysterosystole. Analogous variations in the chronological relations of the waves P and R will be observed, if in place of a decreased discharge through an artery of a cardionector there is an abnormal increase. We shall be dealing no longer with the combination of a systole and a hysterosystole, but of a systole and a proiosystole.

The combination of a proiosystole of the auricle and a ventricular systole results in an elongation of PR , and that of an auricular systole and a ventricular proiosystole results in a shortening of PR . In either alternative if, at each ventricular propulsion the same disequilibrium persists, the relation between the auricular contraction and the ventricular will likewise persist.

All the modalities which we have passed in review, where PR has various values, positive and negative, form in reality a continuous series. This series has been divided artificially and arbitrarily, whence the types with

indefinite limits, called sinus, auricular, and nodal. It is difficult to admit that these types form a continuous series, corresponding to the successive functioning of distinct centers. It is easier to understand that they may be only the translation of relative variations of the sanguineous irrigation of the cardioneotors.

We may also observe on the same film this continuous change in the value of PR. In the following case it is experimental. The tracings in figures 104, 105 and 106 are sections of the same film taken from a patient presenting habitually the inverted sequence RP, who was the subject of a study made in collaboration with Dr. Donzelot.¹ Under the action of amyl nitrite, P posterior to R, gradually comes nearer and nearer to R, is consolidated with it, and then gets in front of it, reconstituting the normal PR interval. The action of the drug having been exhausted P goes down stream again, is once more consolidated with R, and then appears as at the begin-

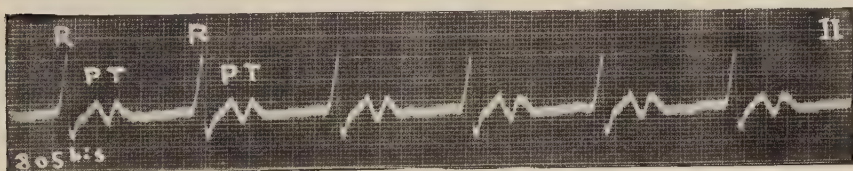


FIG. 108. Permanent auricular hysterosystole due to stenosis of the artery of the atrionector. P follows R, instead of preceding it; the auricle-ventricle sequence is reversed.

ning, after R, and reestablishes the inverted sequence RP. I believe I can now explain this phenomenon as due to an augmentation of the blood supply in the artery of the atrionector whence the shortening of the time of RP. The amount of blood in the artery returns to its usual figure when the vasomotor reaction ceases, RP is lengthened and P resumes its former position in relation to R.

The second tracing in figure 107, taken from another patient, shows a spontaneous progressive variation in the chronological relations of P and R, sometimes in one, sometimes in the opposite direction. The type habitually presented by the patient is the sequence PR. At the moment of the variation, the time of the atrionector RP is prolonged, whence the production of an inverted sequence RP, then a return is made to the normal sequence PR. A momentary diminution in the volume of blood in the artery of the atrionector will well explain this phenomenon. I have been able to furnish anatomo-clinical proof that this explanation is well founded.

CASE REPORT

I had the opportunity to observe in the service of Professor Vaquez at the Pitié, during one year (October, 1924, to November, 1925), a patient whose electrocardiographic tracings were of the type called "atrioventricular rhythm."² He was a man of 63 years, former syphilitic, attacked with aortitis with double murmur at the base and presenting in addition a murmur at the apex. There was constant dyspnea.

The tracings collected from this subject I published in an article appearing in the *Archives des Maladies du Cœur* under the title, "Variations of the chronological relations between the auricular and ventricular contractions." The article appeared in February, 1926, but had been written before the examination of the heart of the subject who died of asystolia November 18, 1925.

In this article I cited the objections that one has a right to make concerning the classical conception of the rhythm known as atrioventricular. I will show in return that it is possible to explain the peculiarities of the electrocardiographic tracings in simple fashion, by taking as a base the hypothesis of cardioneurosis with variable circulation. I terminated the legend of the tracing reproduced at the time with the words, "Pathogenic hypothesis: stenosis of the artery of the atrionector."

The subject of this observation having succumbed in the service, I was able to make an autopsy and to study the heart as completely as possible. The hypothesis which I had formulated was confirmed by the data observed.

The tracing in figure 108 was registered June 29, 1925, and is of the type of permanent auricular hysterosystolia, which would be regarded classically as a type of atrioventricular rhythm of low modality. The tracing published in the article in the *Archives des Maladies du Cœur* was taken on July 8. We made nine other tracings on November 21 and 22, December 21 (1924), March 21, 23, and 30, June 5 and 8, and September 22 (1925). All of these are of the same type, save that registered on June 8, where we see ventricular arrhythmia with auricular tachyrrhythmia of the type of fibrillation.

Autopsy performed November 19, 1925. On opening the thorax a little fluid was found in the left pleural cavity and some adhesions at the corresponding apex of the lung. The right pleural cavity contained no fluid and was free from adhesions. The pericardial sac contained a little lemon-colored fluid.

Right lung: The pulmonary dome was almost entirely transformed into a cavity the size of a small orange, a sort of giant emphysematous vesicle. Other analogous cavities, but smaller, were found in the middle and lower lobes, especially along their anterior border. Along the posterior border there was a pachypleuritis. On section the congested parenchyma was found ridged with bands of slaty sclerosis.

Left lung: Small subpleural ecchymotic spots; toward the apex congestion and edema; emphysema along the anterior border. The upper portion of the lower lobe was transformed into a thin-walled cavity, the size of a mandarin orange, resembling that described in the right lung. On section a little muco-pus from the bronchial cavities could be exuded on pressure, contrasting markedly with the congested background. The pulmonary arteries were more prominent than usual and their section resembled that of the bronchioles.

Liver: Voluminous, weight 1500 grams, on section presenting the classical appearance of the nutmeg liver. The gall-bladder contained a yellow green fluid.

Spleen: Weight 140 grams. Marked perisplenitis with adhesions which made it difficult to remove the organ. The surface was furrowed with numerous fissures. Laid on

the table it did not flatten, and it was of firm consistence. There were several infarcts. The adhesions and constrictions seemed to be connected with the infarcts and consisted of some cicatricial tissue due to loss of substance. Two accessory spleens were found.

Right kidney: Weight 200 grams; strongly adherent and removed with difficulty; adipose capsule slightly developed; small cyst. Surface on section shining and of violaceous color. Malpighian glomerules plainly visible on section. After decortication the surface appeared irregularly depressed, the organ presenting the same fissured aspect as the spleen.

Left kidney: Weight 220 grams; same difficulty in removal because of the adhesions. In the lower pole there was a quite recent infarct, yellow in the center and violaceous at the periphery. The organ after decortication had the same fissured appearance as the right kidney.

Suprarenal capsules: The right was normal and full, the section being firm and the color pearly gray. The left was partly hollowed by a cavity.

Thoracic and Abdominal Aorta: Numerous zones of disseminate endoaortitis along the entire length of the vessel, more abundant and in part ulcerated for a distance of 10 centimeters above the bifurcation.

The Heart: The heart was very large, the hypertrophy affecting especially the left ventricle. The aorta, seen before opening, appeared considerably enlarged and thickened. The sinuses of Valsalva were dilated and formed in their entirety a sort of crown surrounding the base of the aorta. The two coronaries arising from the sinuses were very voluminous, both trunk and branches; they felt hard and thick, but sections at several points of their course showed that the lumen was but slightly narrowed. They were in fact much larger than normally and the diminution of calibre due to parietal thickening was compensated for largely by the extreme size of the entire artery.

On opening the left ventricle, the mitral valve was seen to be thickened and stenosed, and along its lower border it bristled with numerous pointed elevations like spines. The aortic sigmoid valves were thickened, hard, retracted, irregular, with very deep sinuses of Valsalva. But the coronary orifices were not narrowed. In following the aorta from the heart one noted irregularly thickened plaques in its wall, prolonged into the arch and beyond.

The left auricle was increased in volume as was also the right.

The right ventricle presented no peculiarity worthy of note. The region of the cardio-nectors was removed for histological examination.

EXAMINATION OF THE ATRIONECTOR

All of the auricular region corresponding to the atrionector was cut into serial sections made parallel to the interauricular septum, therefore following the long axis of the atrionector. The ribbon comprised 2060 sections, each 7 microns in thickness. One section in 20 was removed and stained, either with hematein (van Gieson), fuchsine, or more rarely with Mallory's mixture.

The atrionector was not the seat of any lesion; at most there was a slightly greater abundance of connective tissue fibers, colored red by fuchsine. The artery of the atrionector here had a normal lumen and walls (fig. 109).

Artery of the Atrionector

This examination was difficult. It is easy to find the artery of the ventriculonector, which, whatever may be its origin from one or the other coronary, has always the same terminal course in the septum which it reaches by



FIG. 109. The atrionector in a case of permanent auricular hysterosystole. The atrionector is divided in its long axis. Ep, epicardium: En, auricular endocardium. The lumen of the artery, AAN, is of normal size and is filled with blood cells. Except for a slight degree of sclerosis, which accentuates the contrast between the auricular myocardium and the atrionector, the latter is normal.

the posterior border; but it is difficult to identify the artery of the atrionector. We know well its termination, but its origin and course vary. We cannot always find the artery immediately. Thus a first cautious dissection

showed the presence of an anterolateral auricular artery given off from the right coronary, 3 cm. from the origin of the latter. This vessel ascended on the lateral wall and corresponded well to one of the known types of artery of the atrionector. But the vessel disappeared quite rapidly in the auricular tissue and I had to give up following its course by dissection or run the risk of spoiling the preparation. I would add that the artery was directed



FIG. 110. The artery of the atrionector projecting into the cavity of the right auricle. AAN, artery of the atrionector; Ep, auricular epicardium. Between the cavity of the auricular appendix, α , α , and that of the atrium, O, is the myocardial mass of the crista terminalis.

forward in such a manner that one would have taken it to be merely a parietal artery supplying the anterior auricular wall.

I then decided to study the sections beginning with the atrionector, in which the artery appeared clearly, and so to reconstitute its course upstream from the atrionector to the coronary; but such a pursuit could not be carried out save by the long and delicate method of serial section. A new block was cut up into 2100 sections and one section out of every 20 was stained and examined.

This reconstitution of the course was quite tedious and difficult, but it enabled me to establish the curious fact which I will relate and which I have never seen mentioned. The artery of the atrionector, at some distance from the latter was seen crawling along the internal wall of the base of the auricle. It was there, almost naked, lodged between the columns of the cavernous tissue of the auricle. Figure 110 shows this peculiar disposition which recalls well enough that of a pulmonary arteriole in a tuberculous cavity, at the beginning of the formation of a Rasmussen aneurysm. In this part of its course the lumen and walls of the artery of the atrionector were normal. In following it step by step I found that this was really the artery of the atrionector. Figure 111 shows schematically its reconstituted trajectory.

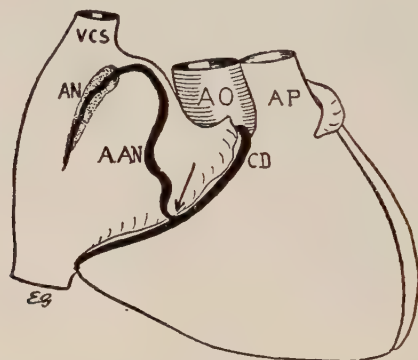


FIG. 111. Schematic representation of the artery of the cardionector. AAN, artery of the atrionector, arising from the right coronary, CD, and extending to the atrionector, AN. The arrow points to the site of the stenosis.

An attentive examination of this artery at its origin from the coronary showed that at this level the vessel seemed to have a narrower calibre, and especially to be rounder and harder. A new block of tissue comprising the coronary and the origin of the artery, lodged in the adipose tissue which filled the auriculoventricular groove, was cut into a ribbon of 480 sections, one in twenty of which was removed and stained. The first examination showed at once that there was an almost complete obliteration of the artery of the atrionector at its origin from the right coronary (figs. 112 and 113). There were 160 sections showing the coronary and the artery of the atrionector. All of these sections, from nos. 121 to 162, or 42 successive sections, were examined, in order to determine with precision the extent and degree of the stenosis. This was very marked and allowed only a narrow

channel of communication between the coronary and the artery of the atrionector. This channel, which could be seen only on the sections numbered 135 to 142, or 8 sections in all, measured only 8 times 7μ , that is 56μ —a little more than the twentieth of a millimeter. In the rest of its course where it was least contracted, the artery measured one-eighth of a millimeter. Even allowing for the retraction of the tissues under the action of the hardening reagents, the stricture was an extremely tight one.

The stenosis was due to a cuff of localized endarteritis just at the origin of the artery of the atrionector and appeared to be a prolongation into this artery of a considerable endarteritis which affected especially the coronary artery and lined it throughout its entire course. The two illustrations are more explicit than any description.

THE VENTRICULONECTOR

The ventriculonector was cut into 2014 serial sections. No lesions were found and the only thing to note is that the septum on which it was seated was thickened, covered on both sides by a thick layer of inflammatory tissue of new formation. In the interior of the septum, too, the connective tissue was swollen in places by inflammatory nodules, which pressed against the ventriculonector at intervals and rendered its course sinuous.

This lining of inflammatory tissue on the endocardium of the septal region corresponded to the inflammatory layer on the aorta, the sigmoids, the coronaries, and also the endocardium of the mitral orifice. It was the same process generalised over the heart and all its vessels. The splenic and renal infarcts above described were due to corresponding foci of endarteritis, as was verified histologically.

Artery of the Ventriculonector

The multiplicity of foci of endarteritis made it necessary to examine this artery. It originated from the same right coronary. At 2 cm. from its origin it also was stenosed but still permitted a sufficient flow of blood, for the rhythm of the ventriculonector was not slowed.

Variations of the chronological relations of P and R do not result either from the activity of the accessory centers situated below the sinus center (shortening of PR) or from the obstacle to the conduction of a stimulus leaving the sino-auricular node and directed toward the ventricle (lengthening of PR).

These variations are due solely to a change in the circulatory equilibrium of the atrionector and ventriculonector.

When the blood supply from the arteries which nourish the cardionections

is uniform the cardiac mechanism is normal; there is a normal precedence of the auricle over the ventricle. If the supply from one of the arteries is more or less modified, the equilibrium is broken and P and R take different positions in relation to each other.

The difficulty which we have found in precisely determining a normal value for the dissociation of PR and the necessity of speaking solely of an average value, is in accord with the hypothesis of an equilibrium between



FIG. 112. Section of the right coronary artery and the origin of the artery of the cardionector. The cuff of endocarditis which lines the coronary extends into the initial portion of the artery of the atrionector, appreciably narrowing its caliber.

two circulations which control, respectively the activity of the auricular and the ventricular cardionectors. On the other hand, if PR represented the conduction time between auricle and ventricle, the very considerable variations of this conduction time, PR, in normal subjects would be something quite unusual.



FIG. 113. Detail of the arterial lesion. This figure corresponds to the enclosed portion of figure 112, greatly magnified. Below is the lumen of the right coronary artery, CD. AAN, artery of the atrionector. The internal elastic membrane, or tunica intima, *me*, fairly well preserved, marks the limit of the original lumen of the artery; all of the materia to the inside of this membrane is newly formed endarteritic tissue which, in the section here shown, completely obstructed the artery of the atrionector. N, N, nerves. Section 134, enlarged 60 diameters. The schematic representation of the artery (in the upper left hand corner of the figure) shows that the artery was patulous between sections 135 and 142 only. A, thickness of the normal arterial wall, limited by the intima, *m*. B, endarteritic new-formation producing the stenosis.

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¹ Donzelot, E., et Géraudel, E.: Un cas de bradycardie septale permanente. Arch. d. mal. du cœur, July, 1924.

² A résumé of this observation was given in a paper read by me on June 20, 1926, before the Académie de Médecine de Paris, entitled "Inversion de la séquence oreillette-ventricule," and the paper was published in full in La Presse Médicale for November 6, 1926, under the title "Un cas de rythme 'atrioventriculaire' (hysterosystolia auriculaire permanente)."

CHAPTER VIII

ANOMALIES OF THE CARDIAC MECHANISM MARKED BY ANISORHYTHMIA

When the rapidity of one of the auricular or ventricular beats ceases to be equal to that of the other beat, there is *auriculo-ventricular anisorhythmia*.

We shall arrange the anisorhythmias in four different groups according as they are due to (a) auricular tachycardia, (b) ventricular tachycardia, (c) auricular bradycardia, and (d) ventricular bradycardia.

When one of the two rhythms departs but little from the mean, it is rated as a basic rhythm. By comparison with this rhythm the modified rhythm is classed as tachyrhythmia or bradyrhythmia, and these modifications are absolute.

But when both rhythms are separated from the mean, there is no longer any basic rhythm and we are obliged to agree on a convention, choosing as basic rhythm that one which is separated least from the mean. By comparison with this basic rhythm, the other rhythm will be considered as the modified one, and characterised as a relative tachyrhythmia or bradyrhythmia.

ANISORHYTHMIA DUE TO DISCORDANT AURICULAR TACHYCARDIA (FLUTTER)

The lower tracing of figure 114 will serve as the type for the description of anisorhythmia due to discordant auricular tachyrhythmia. The P waves succeed one another with a rapidity 180 per minute, while the speed of the R waves is 60. The rhythm of the R being taken as basis, there is auricular tachyrhythmia.

The Auricular Complexes

As the ventricular complexes are here less numerous than the auricular ones, certain of the latter are not covered by the ventricular complex. Thus differentiated they are seen to their full extent and we can appreciate their form. The P wave is only the initial portion; in the curve taken as type, P is quite high, acuminate, and clearly diphasic. The tracing after P presents a double wave which is here but slightly marked, extending as far as the succeeding P. This double wave recalls, on a small scale, that which follows the ventricular complex, and with Hering we will designate by the letter T₂.

If we ignore the ventricular complexes, the curve is made of a line which

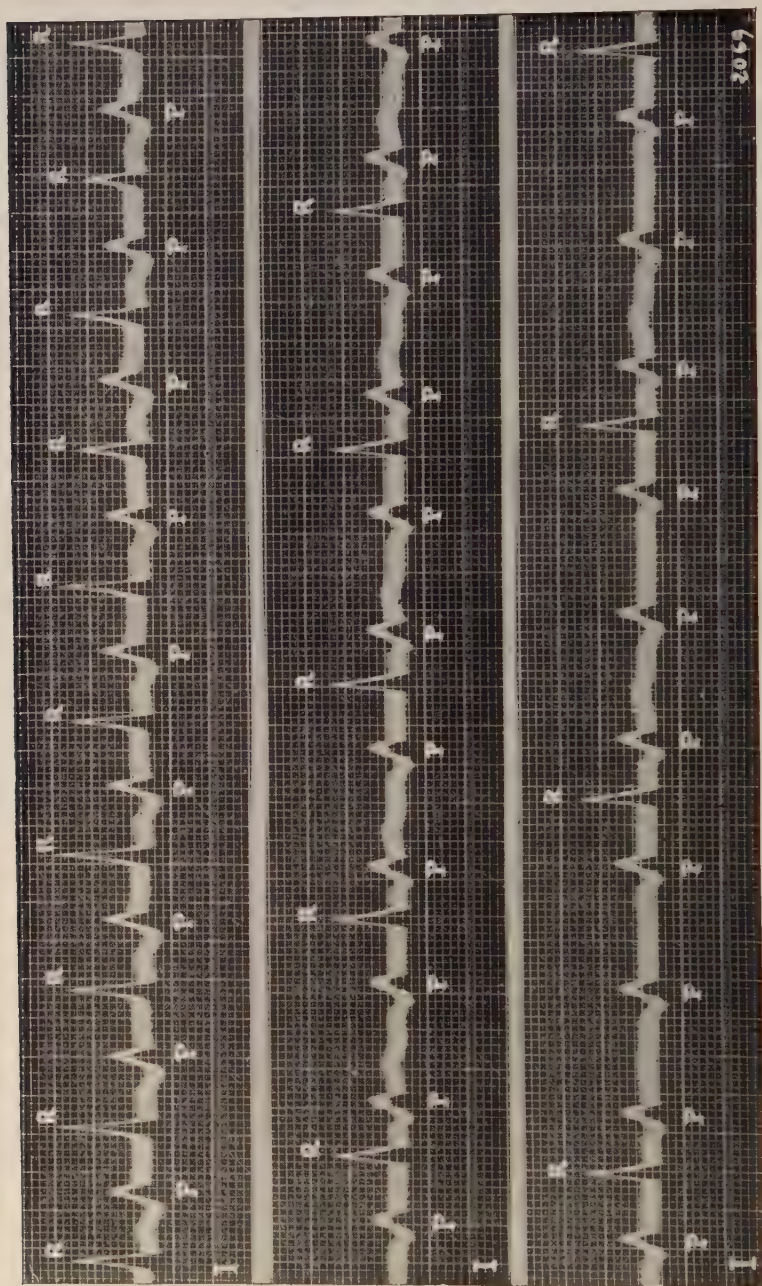


Fig. 114. Anisorhythmia due to discordant auricular tachyarrhythmia ("flutter"). The three tracings are parts of a film from the same subject during the same séance, and all in Lead I. The rapid auricular rhythm is 171, 200, and 189 in the three successive tracings. The ventricular rhythm varies, being 171 in the upper tracing, 100 in the middle, and 63 in the lower. Each of the rhythmic ratios, 171:171, 200:100, 189:63 occurred without any relation to the others.

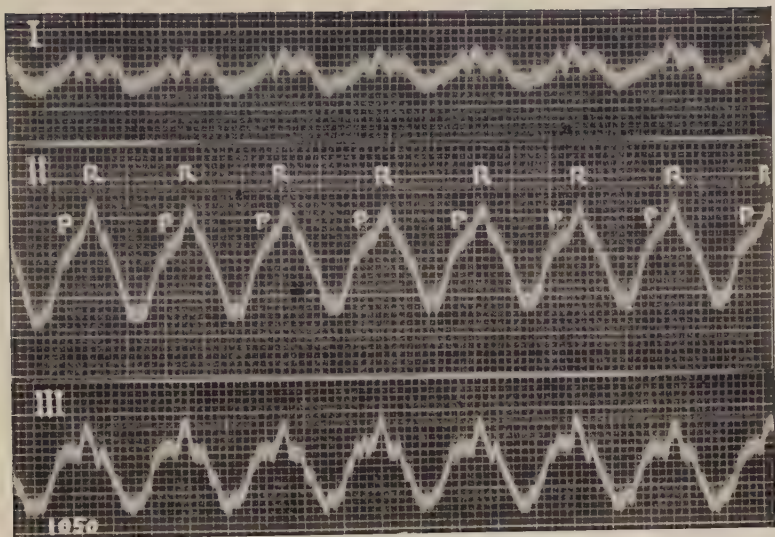


FIG. 115. Anisorhythmia due to discordant auricular tachyrhythmia ("flutter") in the three leads. Ratio 234:234.

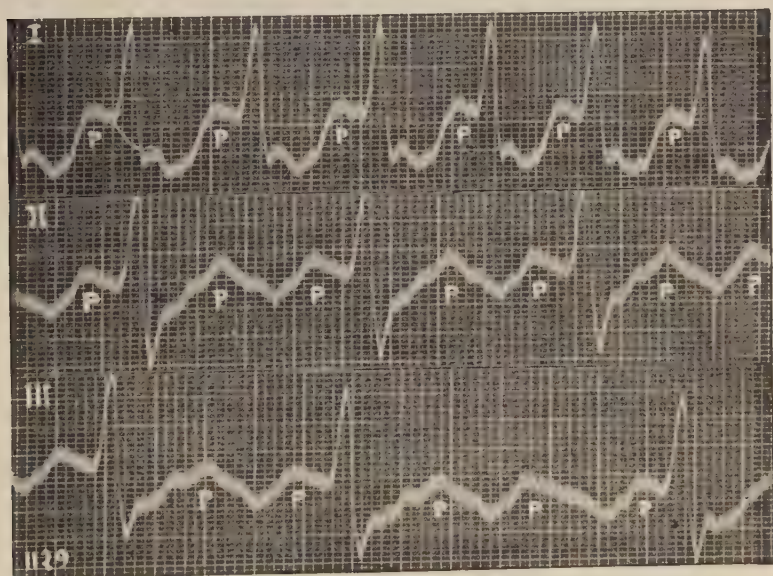


FIG. 116. Anisorhythmia due to discordant auricular tachyrhythmia ("flutter"). In Lead I the rate is 240:240. The dotted line marks the end of the first auricular electrogram. In Lead II the ventricular rate falls to 120. There is auricular allorhythmia, the auricular electrograms P and P are 0.19 second apart, P and P 0.20 second, and so on. In Lead III the gait of the auricular series is the same, but the ventricular rhythm is wholly irregular.

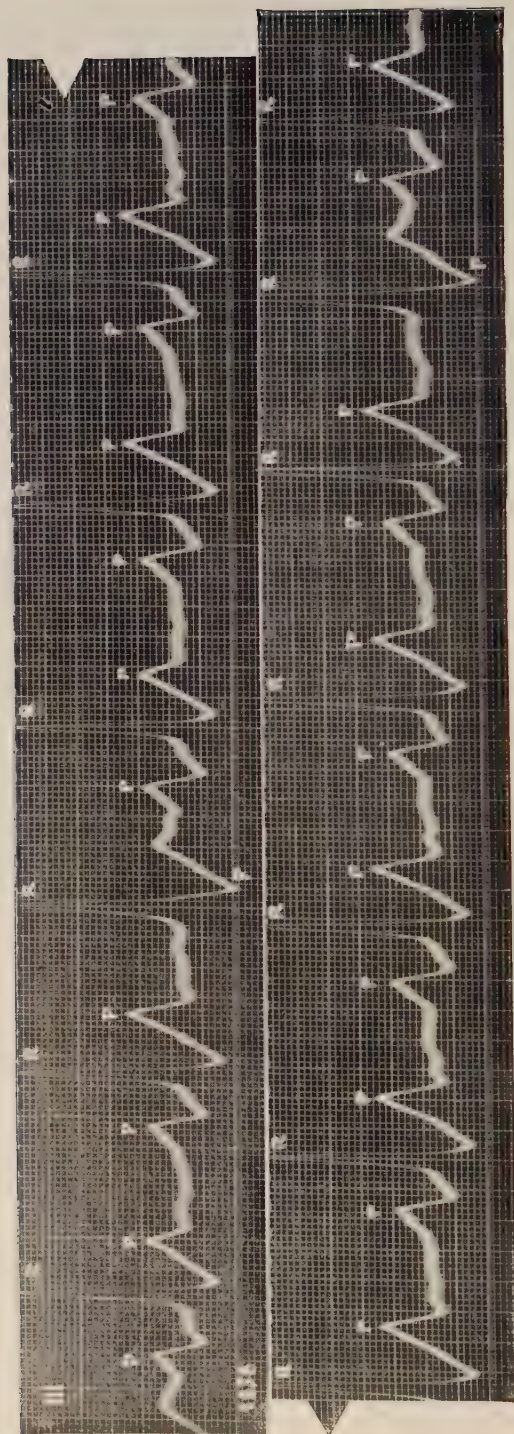


FIG. 117. Anisorhythmia due to discordant auricular tachyarrhythmia ("flutter"). The auricular rate is 160, but it is broken twice by a proisystole. The proisystolic complex is deformed by a negative P wave which prolongs the descending limb of R. It will be noted that the regularity of the auricular rhythm is itself broken curiously, the interval between two successive P waves being regularly shorter by one one-hundredth of a second when a ventricular systole, indicated by R, is interposed between them.

is almost isoelectric, bristling with the waves which are well individualized, recalling the prolongation of the snout of the sawfish. This type of tracing is relatively rare.

Most frequently the auricular complex has a different appearance. (See the different types represented in the illustrations in this chapter.) The two phases of P are very marked; and the summit of the positive phase of one P and the hollow of the negative phase of the P which follows are joined by a more or less obliquely descending line. The result is that the tracing in its entirety quite reminds one of a saw with the teeth in juxtaposition and not isolated as before. The teeth have a variable form and are usually asymmetric, the positive P wave rising above the line being inclined upstream toward the beginning of the film. At times, however, there is greater symmetry. The tip of the positive P wave exactly capping the tooth of the saw. On certain tracings the tooth is depressed and the curve assumes a regularly undulating appearance quite suggestive of the sinusoidal curve. In other cases the tooth is very high and the curve assumes a zig-zag shape (fig. 115). All of these aspects, except the first, may be compared to a graph of the flapping of the wings of a bird, whence the term "flutter" which English authors have given to auricular tachyrythmia.

The portion of the tracing which descends from the positive P wave to the negative P wave following is habitually marked by a secondary wave, and at times it actually bristles with several of these undulations.

The auricular rhythm is most often regular, but the auricular complexes may follow one another in couples, each double pulsation being separated from the double beat which follows by an interval which is greater than that which separates the two events of the couple (fig. 116).

The tracing in figure 117 is remarkable in several of its details: the auricular pulsation is but slightly increased, about 160, while the ventricular rate is 80. This ventricular wave on the tracing is interrupted at irregular intervals by a proiosystole of normal complex, despite its appearance, the apparent deformity depending on the fact that the negative P wave, below the line, lowers the hook S. But the most interesting detail of this tracing lies in the fact that the auricular beat assumes a bigeminal pace, as in the preceding example, but for quite another reason. We note that every time a ventricular systole is inserted between two auricular systoles, the interval between these systoles is shortened. We shall find this peculiarity again in the section on ventricular bradycardia to which we would direct the reader for the explanation which may be given of this occurrence.

The irregularity may be greater and the interval between the two successive auricular complexes may not remain constant. The rapidity of

the auricular rhythm varies; in the examples which we give it may be seen to vary from 160 to 400.

The Ventricular Complexes

The ventricular complexes are habitually of normal form, but the rhythm of the ventricle in cases of auricular tachycardia is extremely variable and is always independent of the auricular rate; each cardiac cavity beats for itself. Sometimes the ventricular beat continues regular, sometimes it becomes irregular.

Interrelation of the Auricular and Ventricular Rhythms

Both the auricular and ventricular rhythms, although of unequal rapidity, are regular, and we may speak of a certain relation between them. This may be simple, as when the quotient of the number of auricular beats divided by the number of ventricular beats in the same length of time is a whole number. It follows from this simple relation that the ventricular beats seem to constitute a fixed group with a certain number of auricular beats.

The middle tracing in figure 114 shows a relation of $\frac{198.0}{84.0} = \frac{2}{1}$ or 2. Once in every two beats P is followed by R (see also fig. 118 where the relation is $\frac{168.8}{84.4} = 2$).

The lower tracing in figure 114 shows a relation of $\frac{168.0}{56.0} = \frac{3}{1} = 3$. Once in every three beats P is followed by R.

The tracings in figures 52, 114 (upper curve), 115, 116, 119, 120, 122, 127, and 128 show a relation of 1 to 1, the velocities being 200 to 200, 180 to 180, 240 to 240, 190 to 190, 298 to 298, 296 to 296, and 168 to 168 respectively. In one case in the same subject, every few seconds or at an interval of months the ventricular beat changes, the auricular beat remaining nearly the same, but the relation of the beats is always a whole number, 1, 2, or 3. It was the same with another patient from whom we took the tracing in figure 52 and the middle and lower tracings in figure 114, the two latter on the same day with a few minutes interval, the first some months previously. This patient showed an auricular pulse almost constantly at 180, while the ventricular beat was 180, 90, 60; P was followed by R, either every time, or once in two or once in three times. As we shall see these facts are explained by a stimulus originating in an ectopic center and putting the ventricle into action after the auricle, either every time, every other time, or every third time, the intercalated stimuli remaining for some unknown reason ineffectual. But in this case the appearance is deceitful, for the interval PR is variable. While the ratio of the beats of auricle and ventricle remains

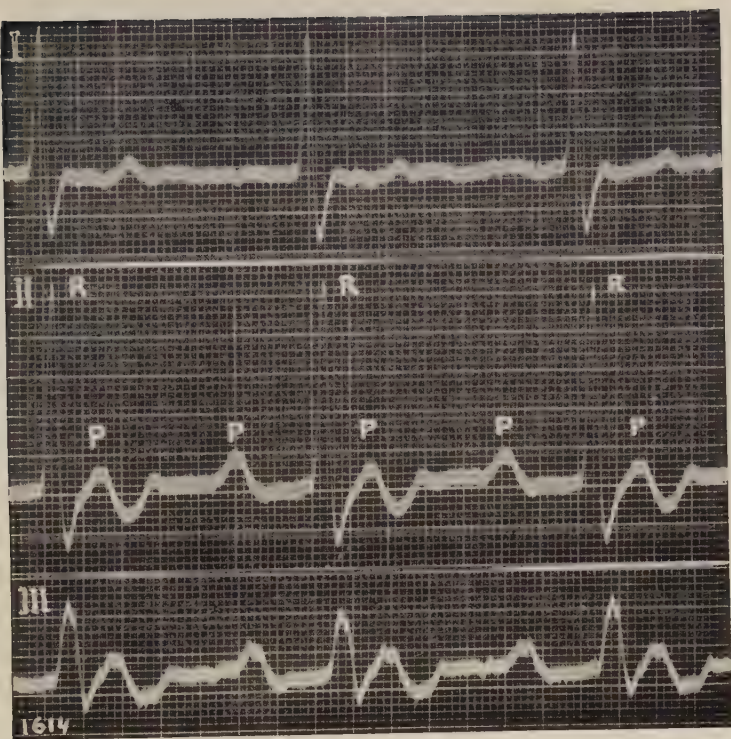


FIG. 118

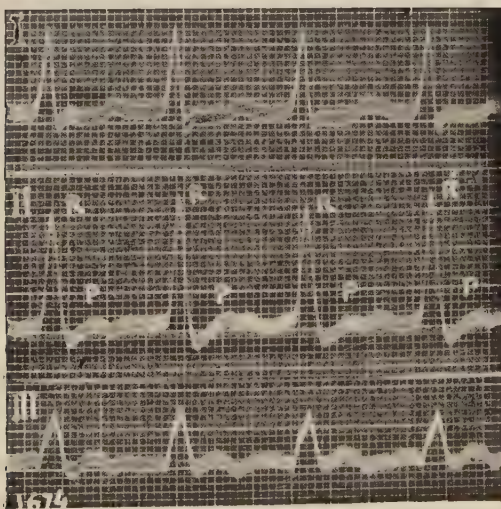


FIG. 119

FIGS. 118 AND 119. Anisorhythmia due to discordant auricular tachyrhythmia ("flutter"). All the tracings belong to the same patient. Those of figure 118 were taken on November 6; those of figure 119 on November 25, 1926. The ratio of the rhythms is 170:85 and 181:181 respectively. It will be noted that the P events in the two figures are completely defaced in Lead I, but are well marked in Leads II and III.

FIG. 120

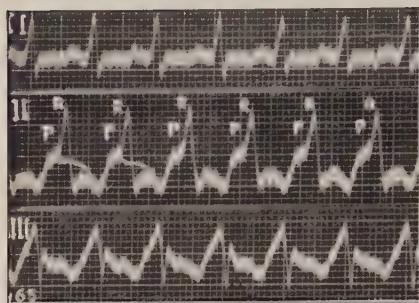


FIG. 121

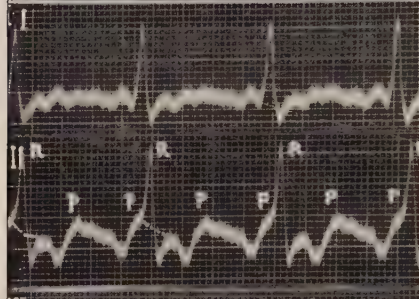


FIG. 122

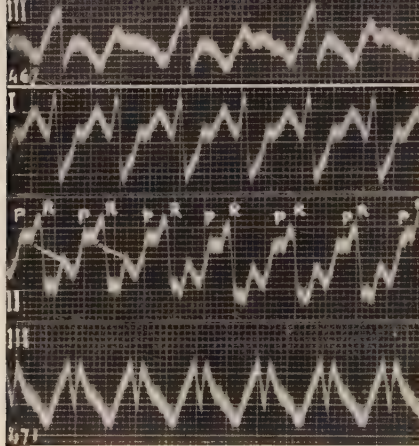


FIG.

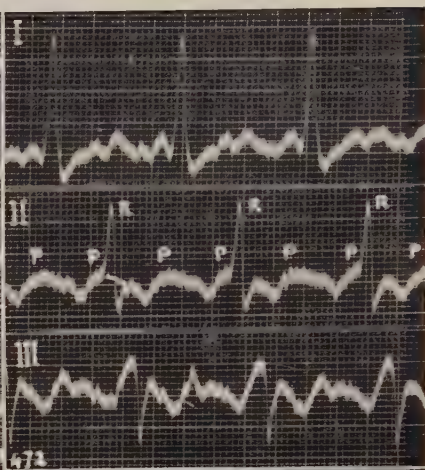


FIG.

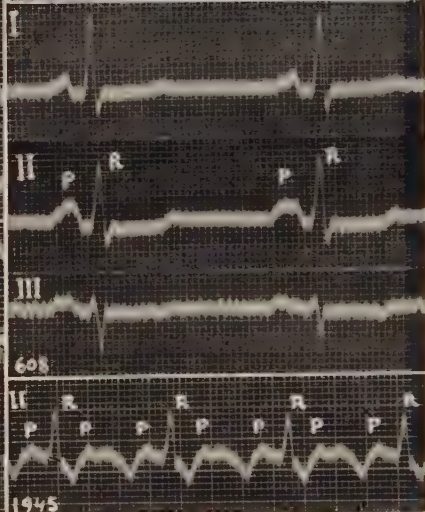


FIG.

FIGS. 120 to 125. Anisorhythmia due to discordant auricular tachyrrhythmia ("flutter"). All these tracings were taken from the same patient, aged 50 years. Fig. 120. February 18, 1925, 300:300—1:1. Fig. 121. February 19, 1925, 298:149—2:1. Fig. 122. February 20, 1925, 298:298—1:1. Fig. 123. Tracing taken a quarter of an hour after the preceding; the ventricular tachyrrhythmia is lowered, 298:149—2:1. Fig. 124. March 24, 1925, return to the normal rates, 80:80, after treatment with digitalis. Fig. 125. February 23, 1927, 320:160—2:1. Note the same peculiarity in figures 120 and 121 as were seen in figures 118 and 119, namely the defacement of P in Lead I, but its very characteristic appearance in Leads II and III.

FIG. 126

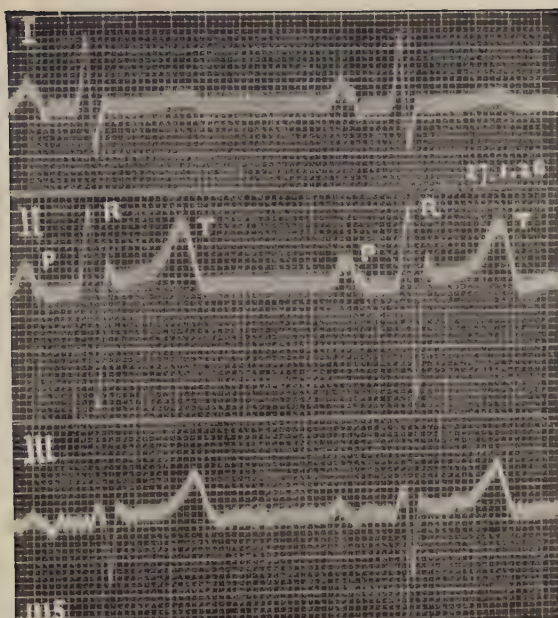
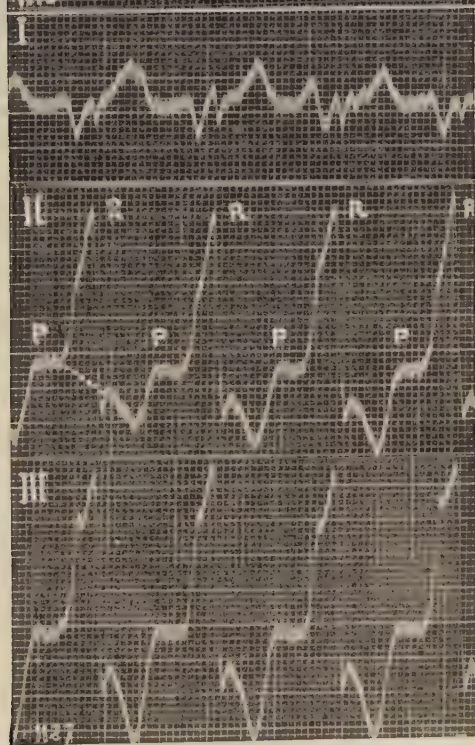


FIG. 127



FIGS. 126 AND 127. Anisorhythmia due to discordant auricular tachyrrhythmia ("flutter"). All these tracings are from the same patient, as were also those in figure 116. The tracings in figure 126 seem to be normal, the rates having the ratio, 65:65. But it is nothing of the sort; a P wave is drowned in the T; flutter exists, but is masked; the real ratio is 130:65. Figure 127 gives the tracings taken two days later during an attack of ventricular tachyrrhythmia imposed on the auricular flutter, the ratio being 166:166. In Lead II the dotted line marks approximately the end of the auricular electrogram.

the same, the PR interval varies greatly. The fact is evident when we compare the curves in figures 121 and 125 taken from the same subject at a year interval. The relation of the rhythms is always 2 to 1, but the auricular wave P, which is nearest to R has its peak blended with that of R in the first tracing, while the two are clearly disengaged in the second tracing. We may also compare with the tracing in figure 52 that in figure 128. The two tracings belong to the same subject. The PR interval, despite the fixity of the ratio of the rhythms, $\frac{200}{200}, \frac{180}{180} = 1$, has a value sometimes of 15/100 sometimes of 17/100 of a second.

The independence of the P and R waves is made evident by these facts, and still more so when the ratio of the rhythms is no longer a whole number.

This is the case in the curve shown in figure 117, in which the formula is $\frac{326}{37} = 9.08$. We note that the position of R in relation to one wave of the curve is extremely variable. We mention this tracing here, for it is clearly

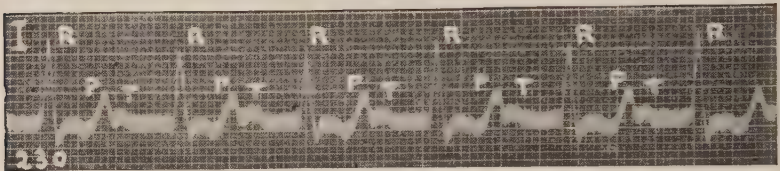


FIG. 128. Auricular tachyrhythmia with the imposition of a paroxysm of ventricular tachyrhythmia; the ratio is 176:176. The last four R events clearly alternate. Compare the relations of P and R respectively on this tracing and on that in figure 52 taken from the same patient from whom also were the tracings in figure 114.

one of auricular tachyrhythmia; but it is also a tracing of ventricular bradyrhythmia so we have placed it in the chapter devoted to this bradyrhythmia. All classifications are artificial.

One may cause a slowing of the ventricle by ocular compression or by compression of the pneumogastric at the neck, but the auricular rhythm is not changed. The curve in figure 129 is a typical example of this slowing of the ventricular contraction. We see that the R waves have no fixed ratio to the P waves.

The P and R waves being independent, their respective positions may vary, as we have seen. When the ventricular tachyrhythmia increases until it equals the auricular tachyrhythmia, the P and R waves, equal in number, may yet be grouped in variable fashion. Sometimes we have the impression that P and R are normally grouped, sometimes P is consolidated with R, and sometimes it follows it. If one remains true to the classical hypothesis, one must conclude that the sequence PR characterizes a "sinusal tachy-



FIG. 129. Influence of ocular compression in auricular tachyrrhythmia. The ventricular rate only is modified, the auricular is not. The arrows and the tablets COC and FC indicate the commencement and the termination of the ocular compression. The interval between the ventricular systoles is indicated by the double horizontal arrows. Tracings from the same patient as that in the preceding figure.

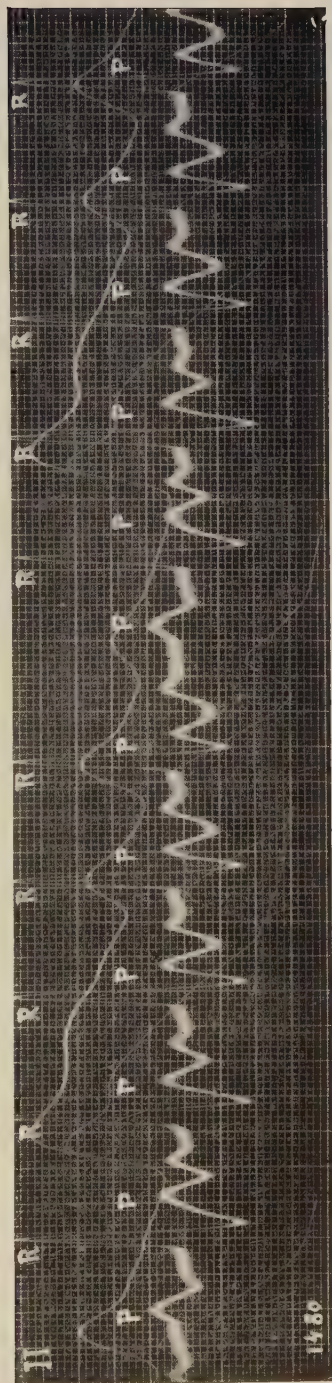


FIG. 130. An episode of ventricular tachyrrhythmia in the course of an auricular tachyrrhythmia. The auricular substratum is revealed between the two ventricular episodes. Had it not been for this interval the entire event would have been diagnosed, according to the classical hypothesis, as a subnodal tachycardia. Both the brachial pulse (lower tracing, faint) and the tibial pulse (upper tracing) are registered. Note the progressive fall of the pulse waves during each of the ventricular episodes and their rise after the ventricular pause.

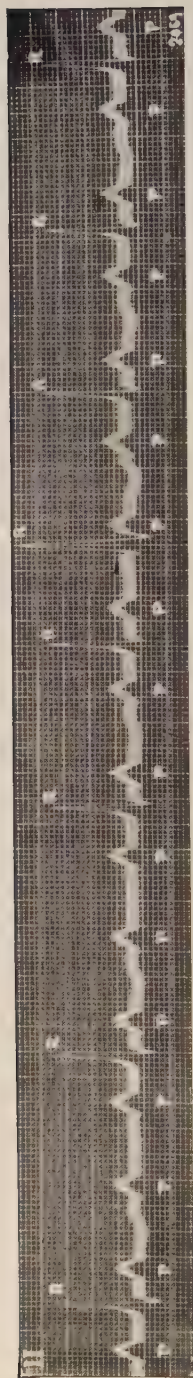


FIG. 131. Auricular tachyrrhythmia. Ventricular rhythm interrupted by a proiosystole

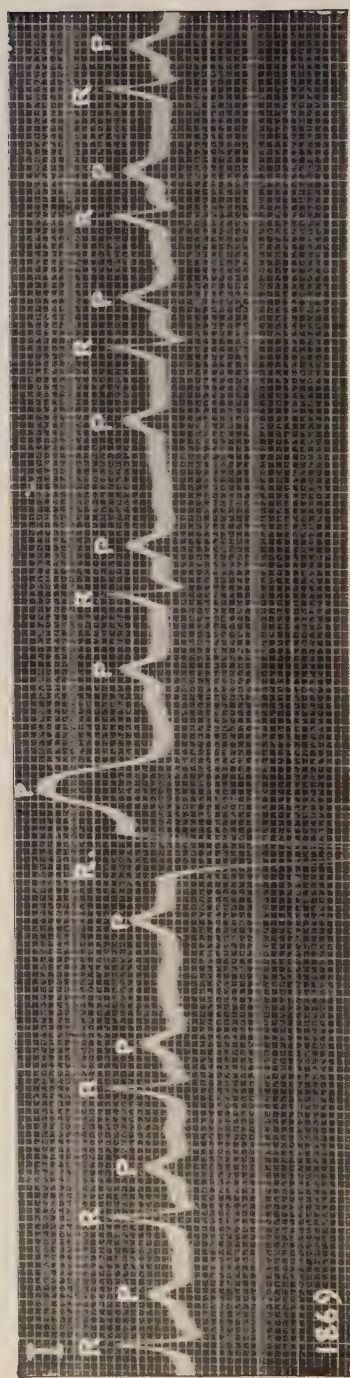


FIG. 132. Auricular tachyrrhythmia. Ventricular tachyrrhythmia broken by a ventricular hysterosystole. The auricular rate is 180 and the paroxysmal ventricular rate becomes the same. There is an alternation of the ventricular complexes. In the middle of the tracing the rhythm is interrupted abruptly by a hysterosystole with deformed complex followed by two hysterosystoles with normal complex; then the ventricular rate returns to 180. The auricular rate continues without break at 180. The tracings in figures 131 and 132 belong to the patient giving the tracings in figures 52, 114, and 128.

cardia;" and that a sequence PR with greatly abbreviated interval, a conjunction of P and R, or an inverted sequence, RP, signifies that there is a "nodal tachycardia" with its varieties—supranodal, nodal proper, and infranodal. It is possible to demonstrate that all this is only apparent.

Let us study, for example, the tracing shown in figure 130. We have here a crisis of ventricular tachyrhythmia grafted on to an auricular tachyrhythmia. This regular auricular rhythm runs from one end of the tracing to the other at a rate of 180 a minute. The ventricular rhythm is manifested by a succession of five systoles at a rate of 180; then there is a ventricular pause, and again the ventricular rhythm is installed at the same rate of 180. The tracing reproduces only the first systoles of the paroxysm.

If we had not, as an aid in deciphering this tracing, the ventricular pause which unmasks the auricular rhythm and if the only documentary evidence we had was the tracing during the paroxysm, we should read RP, RP, etc., and call it infranodal tachycardia. We would imagine that the heart was beating as a whole under the impulsion of a single center commanding both auricle and ventricle. And to explain the inverted sequence RP we should be forced to conclude the existence of a new center, so situated that the stimulus originating there reached the ventricle before the auricle. This center would therefore be situated at a point which corresponds to these special conditions, that is to say, the node of Tawara.

In reality it is not tachycardia but auricular tachyrhythmia and a super-added crisis of ventricular tachyrhythmia.

It is quite useless to seek to localize the phenomenon which causes the contraction of the auricle and the ventricle in centers other than the normal—the atrionector and the ventriculonector.

The auricular tachyrhythmia remaining regular and constant, the ventricular rhythm in place of being regular is irregular and we can no longer speak of a definite ratio between the rhythms. The ventricular rhythm may be irregular because of the occurrence of isolated proiosystoles. This is shown in the curves 131 and 132.

Another type of irregularity results from bigeminal or trigeminal ventricular proiosystoles. The tracings in figures 133 and 134 are two examples of bigeminism. In figure 134 may be seen a regular alternation in the direction of the initial events of the proiosystolic complex. In figure 135 trigeminism is seen combined with bigeminism, and the same is true in figure 136, where the auricular tachyrhythmia has a peculiarly rapid rate of 400. A rather unusual trigeminism is seen in figure 137, the systoles being grouped in threes, but the complex varies with each systole, the two intervals between the three systoles being different; all of these differences are faithfully

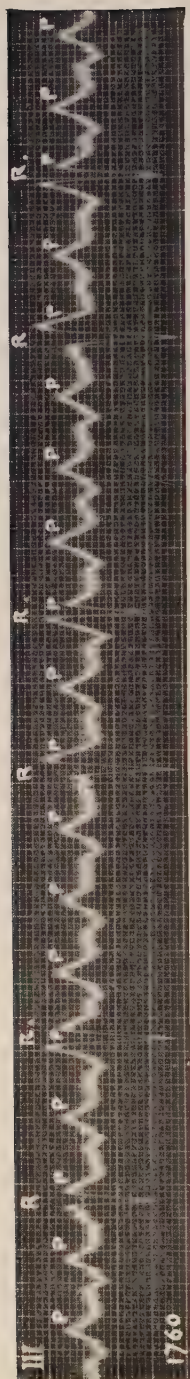


FIG. 133. Auricular tachyrrhythmia. Ventricular bigeminal allorhythmia. From the same patient as the preceding tracing

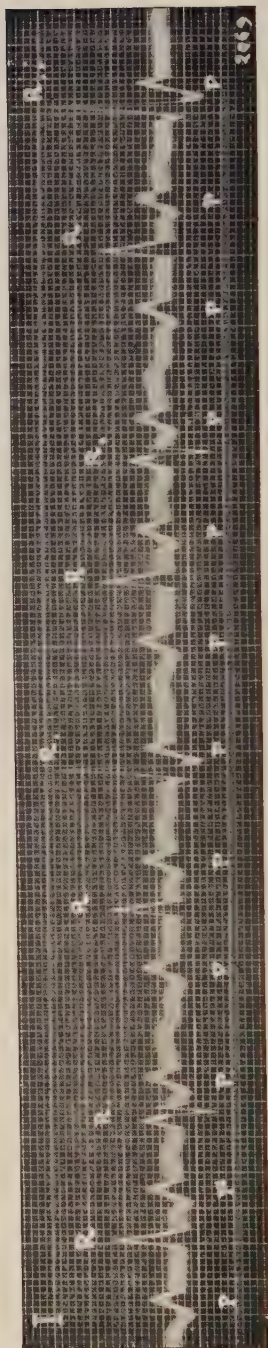


FIG. 134. Auricular tachyrrhythmia. Ventricular bigeminal allorhythmia. The protosystole coupled with the preceding systole is alternately in a positive and a negative direction.

reproduced for each group and in the same order. It will be seen that the interval between the extreme systoles of the group differs but little from the interval between two groups; the tracing can be regarded as the combination of equidistant systoles, which once every two times admit between them a so-called "interpolated" proisystole.

In the preceding types a sort of regularity persists in the irregularity of the rhythm. This may, however, be wanting, as is shown in figure 138; compare also figure 152. It puts well in evidence the independence of the auricular and ventricular complexes. The auricular rhythm is here very rapid—375. The tracing in figure 139 shows an irregular ventricular rhythm, where the complexes are sometimes normal and sometimes not.

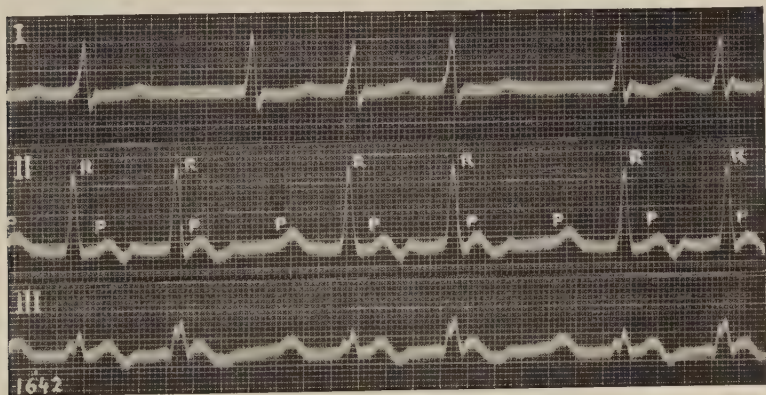


FIG. 135. Auricular tachyrhythmia. Ventricular bigeminal and trigeminal aliorhythmia. In Lead I the aliorhythmia is by trigeminism; in Leads II and III, taken some seconds after the first there is bigeminism. The patient is the same as the one from whom the tracing in figure 130 was taken.

The auricular rhythm is 300. The same auricular rhythm of 300 and the same ventricular arhythmia are seen in the tracing figure 140.

The preceding tracings should be considered as leading up to the facts which we are about to describe in studying the form of hypertachyrhythmia called auricular fibrillation.

Mode of Origin and Termination of the Crisis of Auricular Tachyrhythmia.

In all these cases of discordant auricular tachyrhythmia sudden in the onset of the crisis and sudden in its disappearance, it is interesting to note the way in which the cardiac mechanism acts.

The tracing in figure 70 is particularly illuminating, for it registers the entire phenomenon. The rhythm at a given moment is broken by the

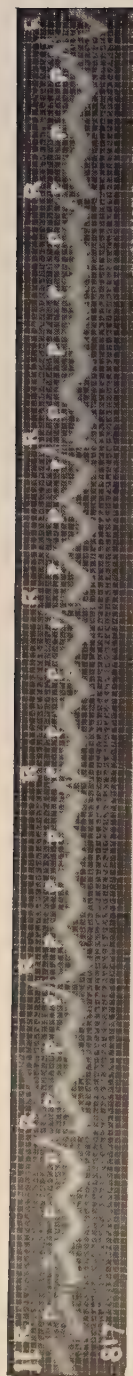


FIG. 136. Auricular tachyrrhythmia. Bigeminal and trigeminal allorhythmia. The bigeminalism and trigeminalism follow each other in the same lead, giving an appearance of irregularity to the ventricular series. The auricular series remains regular; its rate, however, is very rapid, 405. This case may be regarded as a state of transition with auricular hypertachycardia.

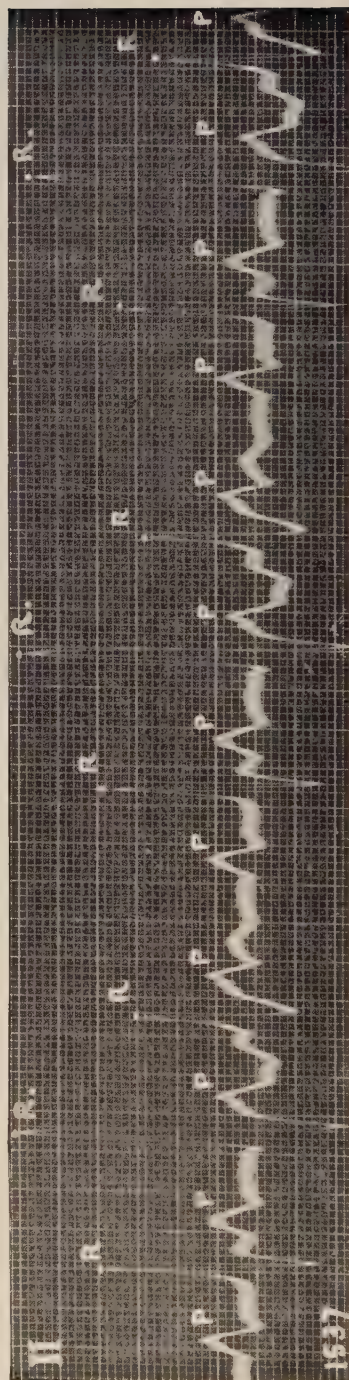


FIG. 137. Auricular tachyrrhythmia. Trigeminal allorhythmia. Same patient from whom were taken tracings in figures 52, 114, 128, 131, and 132



FIG. 138. Auricular tachyrrhythmia. Ventricular arhythmia. The auricular rate is 375. This case may be regarded as one of transition to auricular hypertachycardia. Cf the tracing in figure 148 taken from the same patient

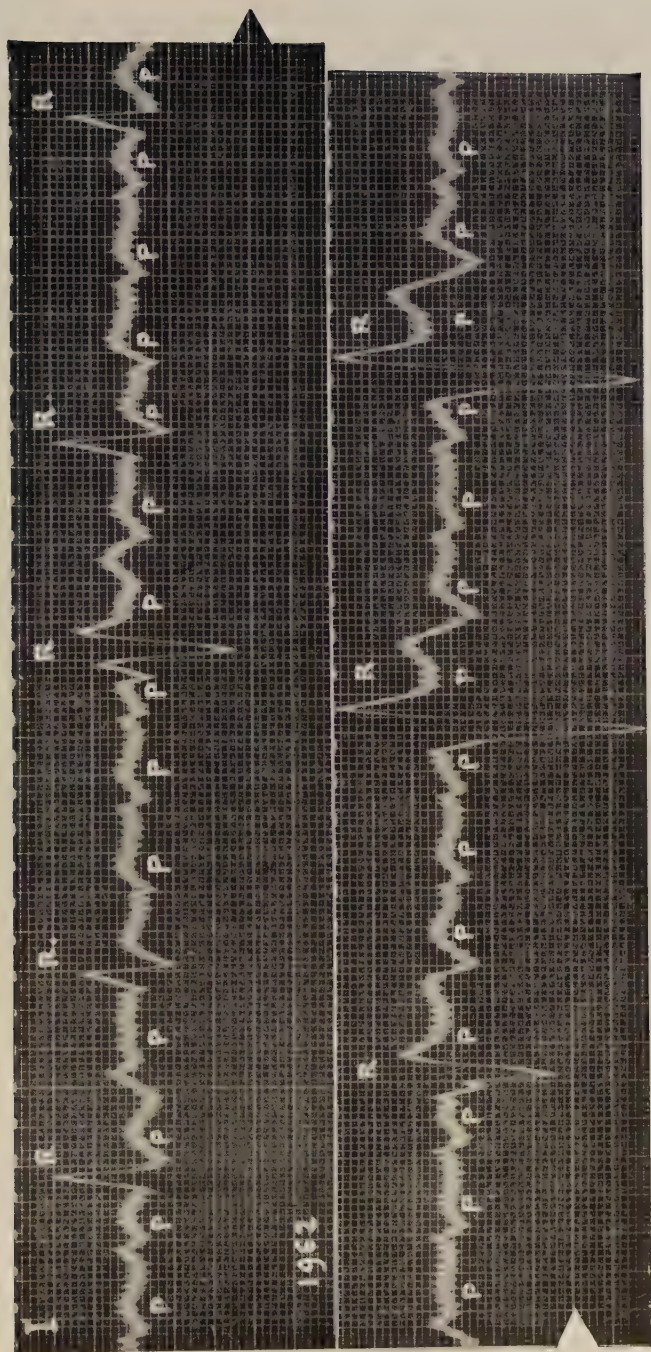


FIG. 139. Auricular tachyarrhythmia. Ventricular arrhythmia. The auricular rate is 300. The ventricular series is very irregular. There is a suggestion of bigeminism in the upper half of the tracing. The ventricular complexes are variously deformed. There is transition to auricular hypertachycardia.

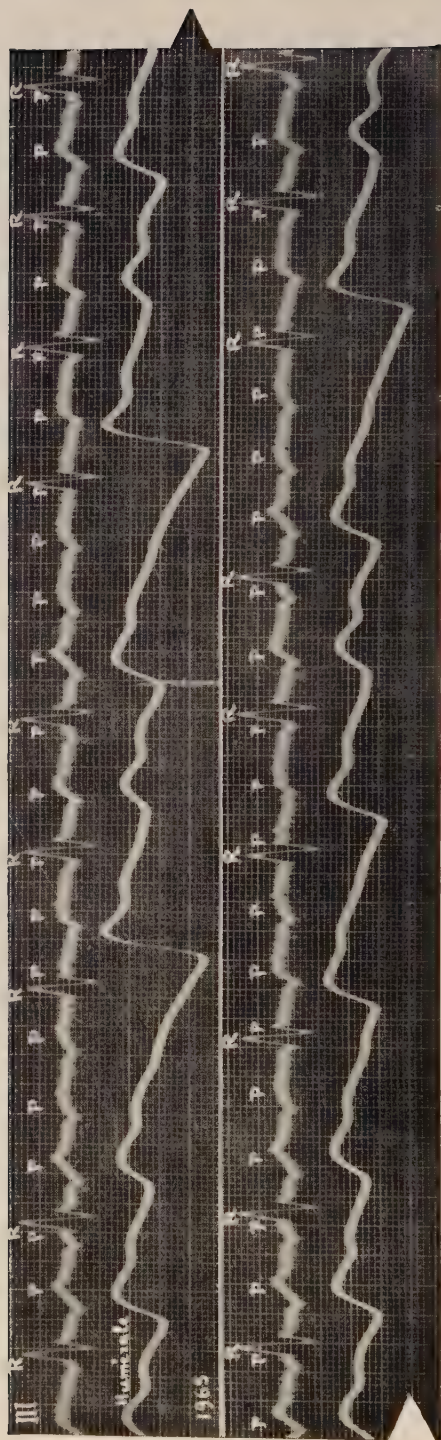


FIG. 140. Auricular tachyarrhythmia. Ventricular arrhythmia. The auricular rate is 300. The ventricular series is very irregular, with a tendency to trigeminism. Note the appearance of the arterial tracing. A case of transition to auricular hypertachycardia.



Fig. 141. The end of an attack of auricular tachyarrhythmia.

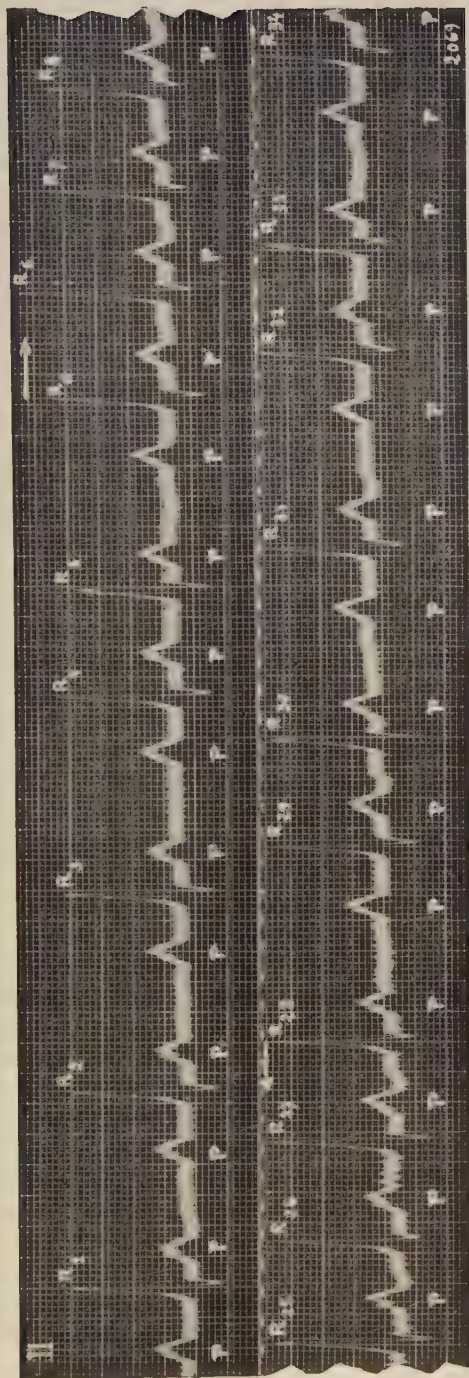


Fig. 142. An episode of ventricular tachyarrhythmia imposed on an auricular tachyarrhythmia. The entire attack of ventricular tachycardia was registered, the two arrows marking its beginning and end, but the intervening part of the tracing is here omitted. It will be seen that the attack begins by a slightly deformed systolic complex, the second R_a (marked by this through error). Comparing this couplet with an analogous couplet in figure 131, taken from the same patient, we see that the ventricular protosystole of figure 131 may be regarded as the fuse of an aborted discharge of tachycardia. Before, as well as after the attack of ventricular tachyarrhythmia, the ventricular series shows a bigeminal allorhythmia in which one must see the prelude and the sequel of the attack.

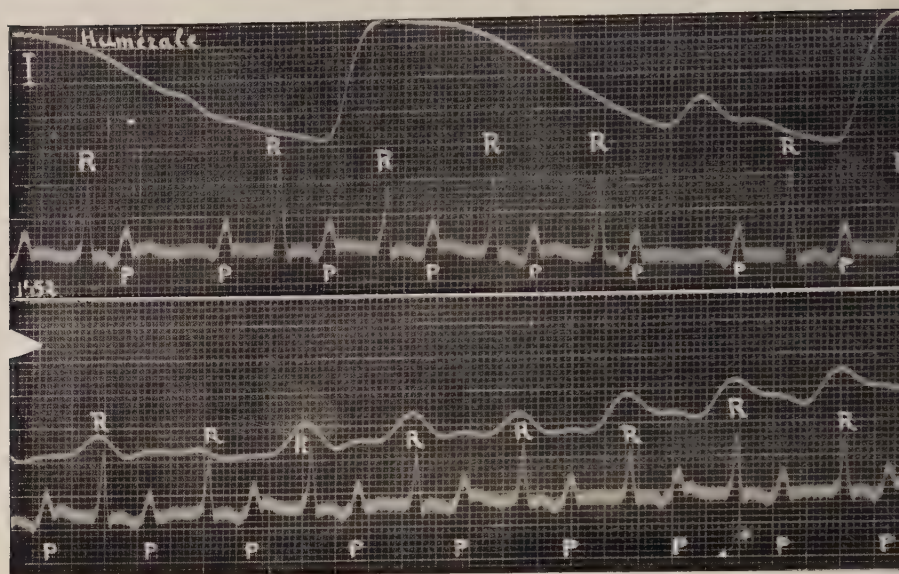


FIG. 143. An episode of ventricular tachyrhythmia imposed on an auricular tachyrhythm. Note the same beginning by a slightly deformed ventricular proisystolic complex, and the same inequalities of arterial pressure—its fall at the moment of the attack, and then its accommodation.

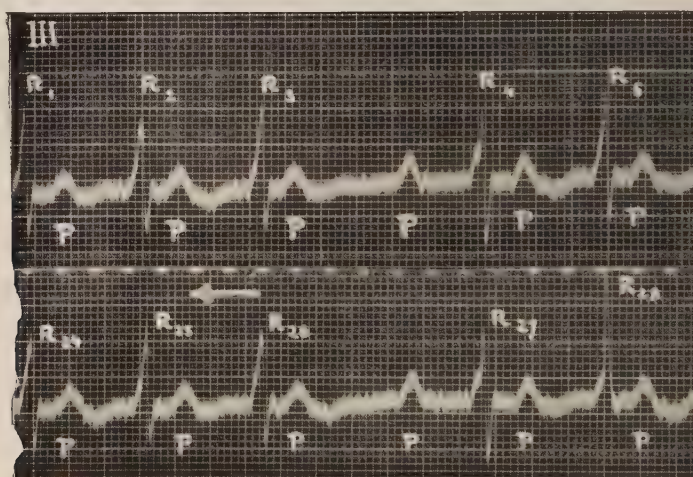
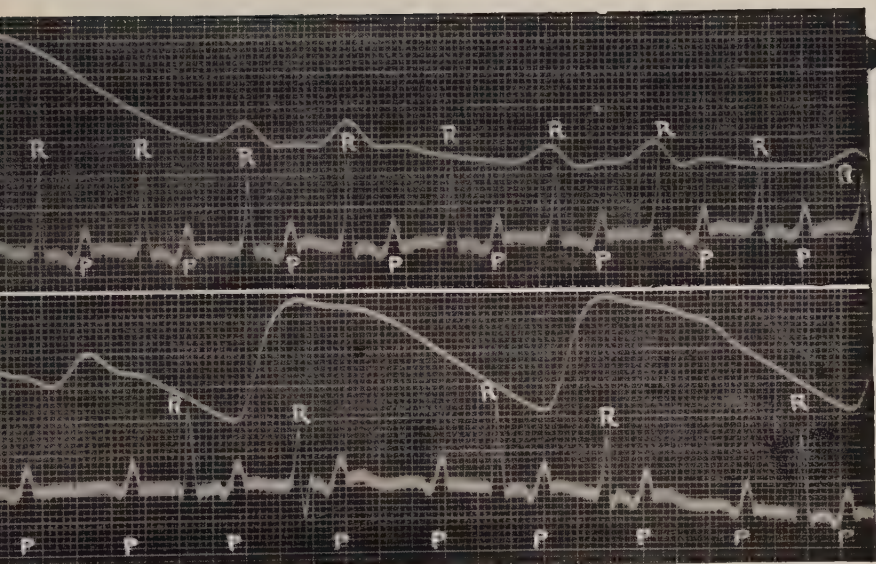
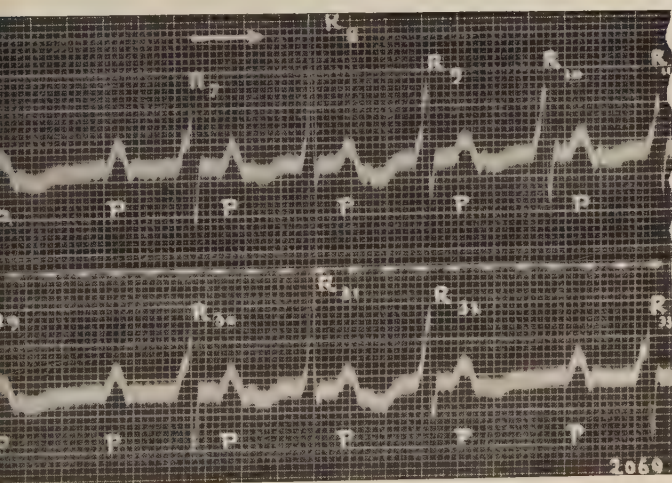


FIG. 144. An episode of ventricular tachyrhythmia imposed on an auricular tachyrhythm as shown. This tracing is from the same patient as those in figures 142 and 143. Note the same beginning by a proisystolic complex. Before and after the attack is a ventricular allorhythmia. We may regard each of these allorhythmic



The entire episode is shown. The tracing is from the same patient as the preceding. We can see the anisorhythmic disturbances occurring before and after the attack. The brachial pulse curve shows the changes.



ar tachyarrhythmia. The beginning and the close only of the attack are shown. The beginning is the same in each case, by a deformed ventricular tachyarrhythmia of trigeminal type. This allorhythmic group reproduces remarkably well the rough outlines of the ventricular tachyarrhythmic attack.

apparition of three proiosystoles—auricular, ventricular, and again auricular. Then the normal rhythm is resumed, to be soon again broken by a new grouping of proiosystoles. Finally the paroxysm of auricular tachyrhythmia is installed, made up of auricular proiosystoles of identical form and succeeding one another at regular intervals. The ventricular complexes also follow one another more or less regularly. At a given moment the auricular tachyrhythmia ceases and after a longer or shorter pause, the normal rhythm is resumed either definitely, as in figure 141, or, as in the curve which we are about to analyze, to be again broken by a proiosystolic group, heralding a new crisis of tachyrhythmia. This group is not reproduced in the figure.

Superposition on the Auricular Tachyrhythmia of a Crisis of Ventricular Tachyrhythmia. We have just seen how the paroxysm of tachyrhythmia is begun and how it ends. The curve in figure 142 shows us how, in the course of a crises of auricular tachyrhythmia, a paroxysm of ventricular tachyrhythmia may appear and again disappear. Before, during, and after this ventricular paroxysm the auricular rhythm remains unchanged at 192. We will for the present ignore the auricular events, so as to study only the ventricular complexes. The basic rhythm of the ventricle is bigeminal; it is found at the beginning and again at the end of the curve. The crisis of ventricular tachyrhythmia is announced by a primary grouping of ventricular beats, a sort of abortive crisis. Between this abortive crisis and the crisis properly speaking the auricular basic rhythm reappears for an instant. It will be noted that the abortive crisis and the beginning of the crisis proper have an identical pace. It is the same with the tracing in figure 143. A ventricular systole is followed by a deformed proiosystolic complex and this itself is followed by other complexes not deformed, succeeding one another at the rate inaugurated by the proiosystole. Two of these undeformed complexes are seen in the abortive crisis, but they are more numerous in the established paroxysm. The rhythm is the same and is equal to the auricular rhythm. The crisis terminates simply. The basic rhythm succeeds to the rapid rhythm, preserving the same bigeminal type which it presented at the outset.

The radial pulse was registered at the same time as the electrocardiogram and the double tracing gives us the opportunity to call attention to the remarkable way in which the arterial pressure behaves. It falls with the appearance of the crisis, the elevations with which the curve bristles being feeble and irregular and fewer in number than the ventricular systoles. Then the curve rights itself, the elevations are more ample and their number becomes equal to that of the systoles. It is as though the cardiovascular

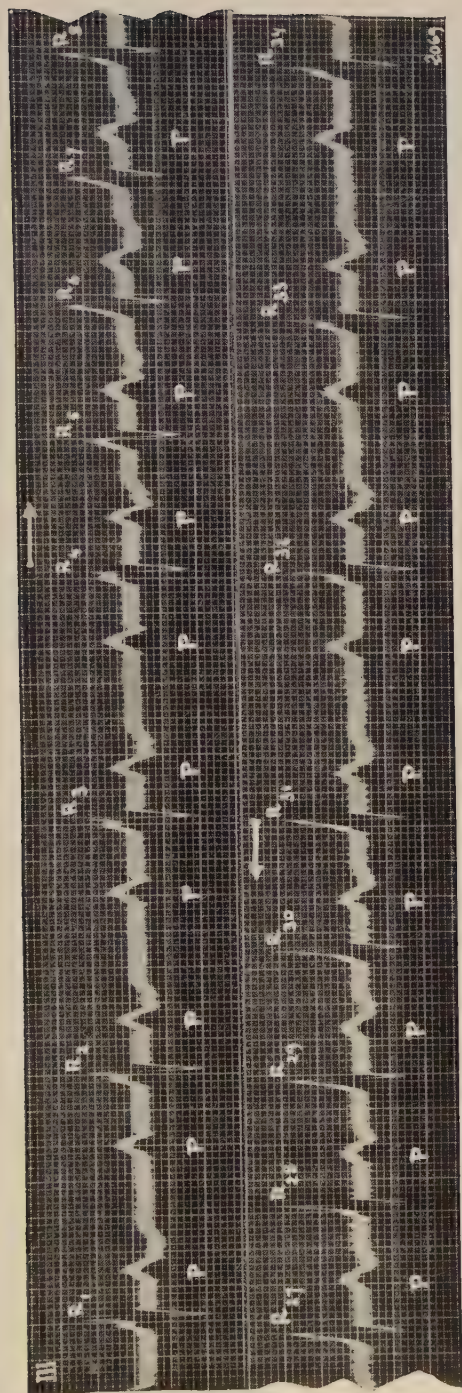


FIG. 145. An episode of ventricular tachyarrhythmia imposed on an auricular tachyarrhythmia. The beginning and the close only of the attack are shown. This tracing is from the same patient as those in figures 142, 143, and 144. Differing from the preceding tracings, the ventricular crisis is neither preceded nor followed by the allorhythmic groups; the ventricular series, both before and after the tachyarrhythmic episode, is regular.



FIG. 146. An episode of ventricular tachyarrhythmia superposed on an auricular tachyarrhythmia. Interruption of the ventricular series by two proisystoles. (This tracing was exceptionally retouched with water colors, its original paleness rendering it scarcely readable.)

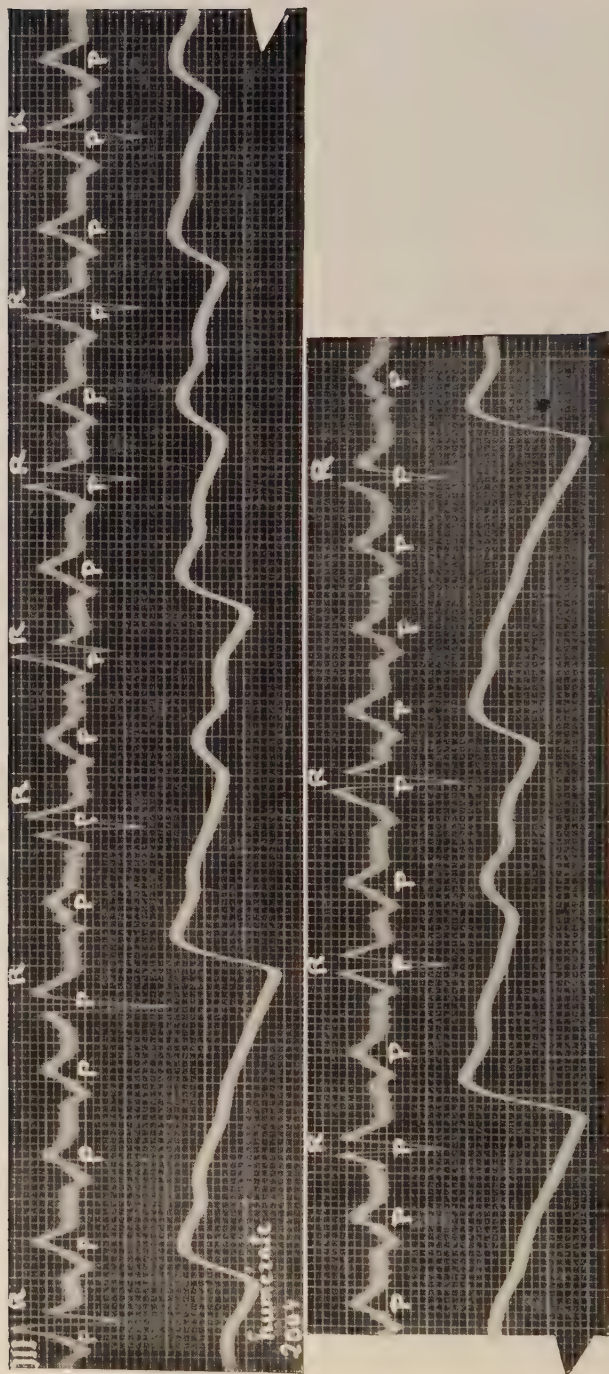


Fig. 147. An episode of ventricular tachyarrhythmia imposed on an auricular tachyarrhythmia. The brief attack of ventricular tachyarrhythmia is reproduced entire; it begins suddenly without any allorhythmic prelude, but is followed by a trigeminal allorhythmic sequel.

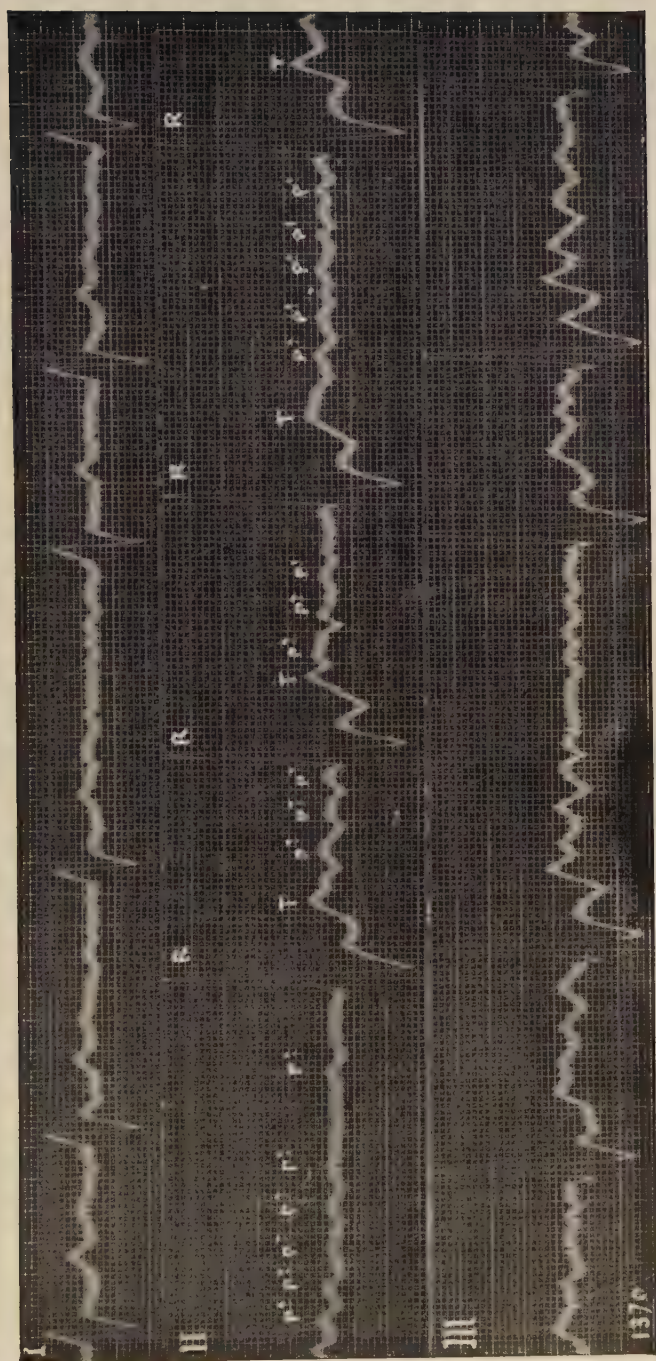


FIG. 148. Anisorhythmia due to discordant auricular hypertachyrythmia (fibrillation). Compare this tracing with that in figure 138 obtained from the same patient nine days before.

system, surprised by the crisis, coördinated the activities of its different parts poorly at first, but later adapted itself to the new régime. We see here a phenomenon of adaptation analogous to that to which attention was called in the section on *Pulsus Alternans* in Chapter IV.

The tracing in figure 144 is another example of a crisis of ventricular tachy-rhythmia grafted on a preceding auricular tachyrhythmia. The crisis is preceded and followed by groupings of the proiosystolic type, of three ventricular beats.

The crisis of ventricular tachyrhythmia may begin abruptly and end sharply without being framed by ventricular disturbances, either before or after (fig. 145). On the tracing in figure 147 there is only one residual group after a short crisis.

During the crisis of ventricular tachyrhythmia grafted on an auricular tachyrhythmia the ventricular series may exceptionally present proiosystoles (fig. 146).

AURICULAR HYPERTACHYRHYTHMIA—AURICULAR FIBRILLATION

In the cases previously cited the rate of the auricular tachyrhythmia did not exceed 300 to 320 per minute, but this figure may be largely exceeded and the rate may attain 500 to 600 beats a minute. This is auricular hypertachyrhythmia. It is classical to treat these facts apart under a special rubric, that of auricular fibrillation, and the denomination of simple arrhythmia is also employed. Auricular fibrillation or complete arrhythmia has, it is said, two characteristics, the first of which is the disappearance of the auricular complex P prior to that of the ventricular complex. The second characteristic is the arrhythmia of the ventricle, but there is nothing specific about that, for it was encountered frequently in the cases of auricular tachyrhythmia already studied. The first characteristic merits a careful analysis. If we ignore the ventricular complexes, the entire tracing bristles with elevations of irregular form, amplitude, and succession (fig. 148). Some of these would suggest the wave P as it appears in the rapid flutters but their rate is higher—from 400 to 600 a minute. Others do not recall P at all being made up of minimal elevations of which the number is near 2000 a minute. They are marked on the tracings by the letter *f*. The two may follow each other in an irregular series on the same curve, which we call impure flutter or fibrillo-flutter (fig. 149).

The tracing of fibrillation should not be confounded with the tracings registering fine muscular tremors. The latter, tremulous in their entire extent, show nevertheless that there is regularly before each R, a P wave, which is easily recognizable although quavering.

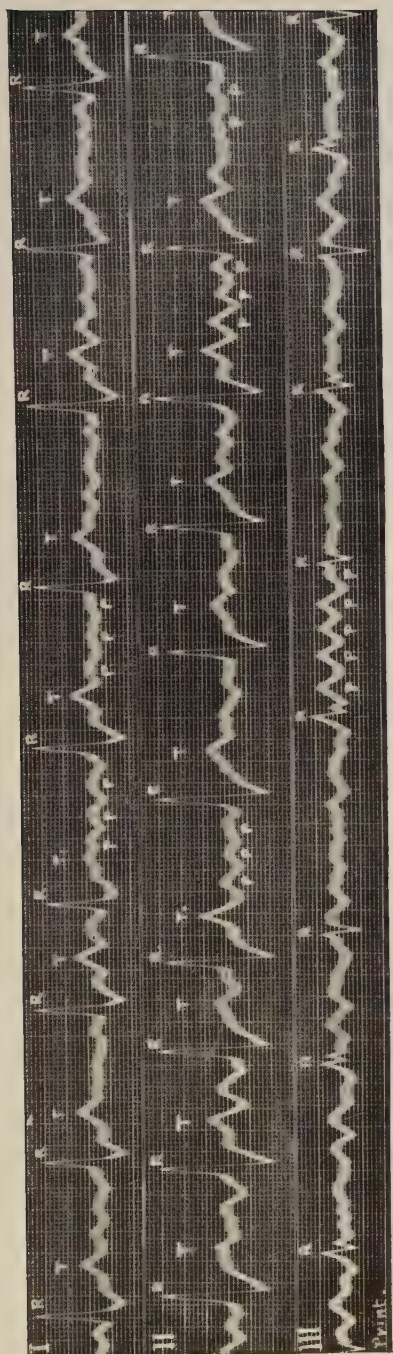


FIG. 149. Anisorhythmia due to discordant auricular hypertachyrhythmia (fibrillation)

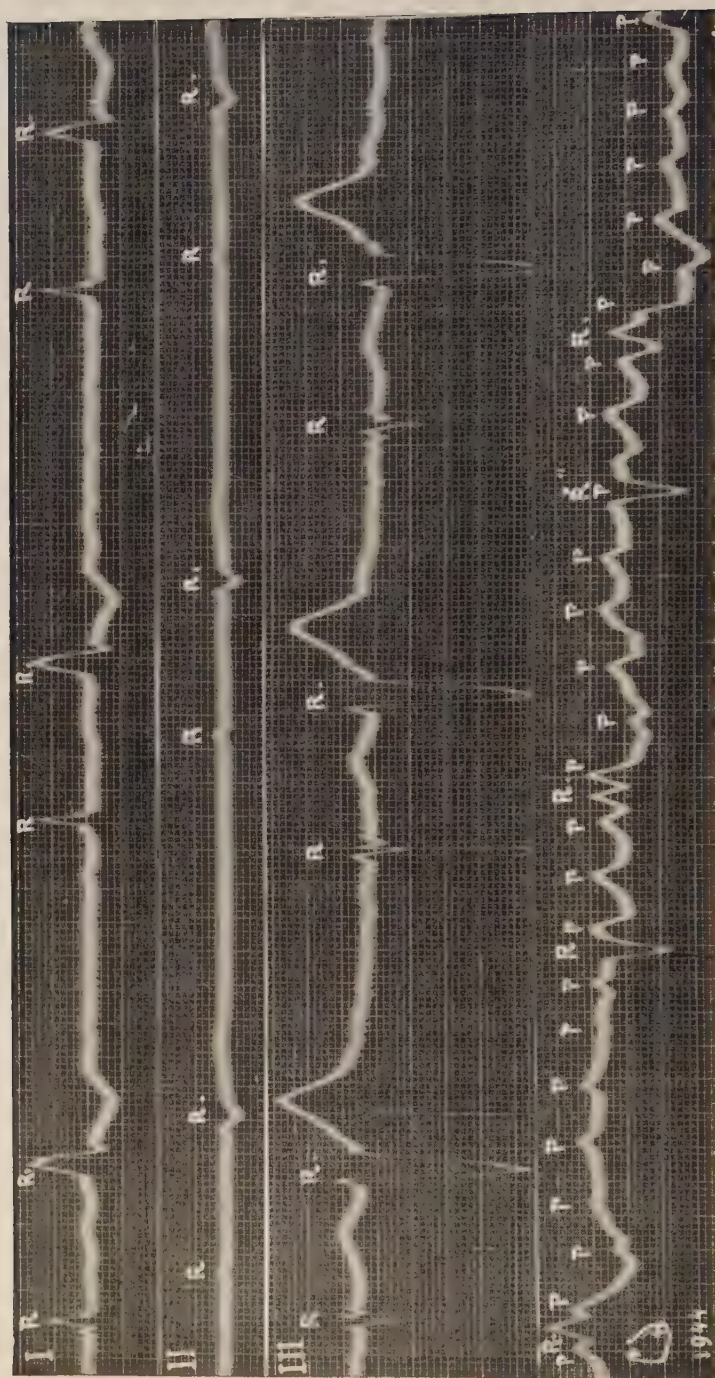


FIG. 150. Anisorhythmia due to discordant auricular hypertachyrhythmia. In Leads I, II, and III, the auricular rhythm is not visible, but it is so in a special lead made by placing the electrodes in the precordial region where marked by crosses in the diagram; as soon as this was done, as shown in the lower tracing, the auricular rhythm appeared. Note the bigeminal ventricular allorhythmia.

The polymorphous events *P* and *f* which cause the bristling appearance of the tracing in auricular fibrillation are but slightly marked in Lead I, but are more accentuated in Lead II and Lead III. They may nevertheless be lacking in all three leads. But if we utilize a lead in which the two electrodes are placed on the thoracic wall along the right border of the heart, or a lead where one electrode is placed on the precordial region and the other on the dorsal region at the same level, the irregular tracing of the fibrillation is manifested, as has been shown by Lewis (fig. 150).

All of these events represent definitely only auricular electrograms, in more or less degraded forms.

This degradation of the form of the auricular electrogram is comparable to that which the ventricular electrogram shows, for example, in connection with the respiratory phases (figs. 50, 51). The tracing in figure 151 shows this auricular degradation produced with a medium rate in a heart functioning normally. The initial event of the auricular electrogram, it may be *P*, is at first of normal aspect, but its form is progressively modified; at the fourth beat it appears fragmented in several unequal peaks which succeed one another at irregular intervals. Then *P* presents a negative phase which increases progressively. At the antepenultimate beat *P* appears again as fragmented, in the same time that its initial phase again becomes positive. The sixteen successive beats of the auricle in this tracing are then marked by sixteen electrograms of different form. If we assume that in place of beating at a rate of 75 to the minute, the auricles beat at a rate of 500 or 600, we would see that the series of auricular electrograms influenced by analogous cause (respiratory phases, vasomotor variations, change in the volume and relations of the cardiac and vascular cavities, etc.) would be decomposed into successive series of electrograms, where *P* would assume one of the sixteen preceding aspects. The succession of several *P* waves of the initial type will give the *P* of the type of the tracing in figure 149 (L. III). The succession of *P* waves of the fourth auricular event will correspond for the rest of the tracing to the irregularities which have been marked by the letter *f*. The "fibrillo-flutter" then is nothing but a mixture of two different modalities of the cardiac mechanism. It is always a matter of only a single modality—auricular tachyrhythmia; the form alone of the auricular electrograms varies and this variation has nothing pathological about it.

In support of this way of looking at it, I submit the two tracings combined in figure 152. These two tracings belong to the same subject who has furnished us already with the tracings reproduced in figures 70, 141 and 151. Both were taken at a few seconds interval, with the same lead and with the same tension of the galvanometer string. The aspect of the ventricular

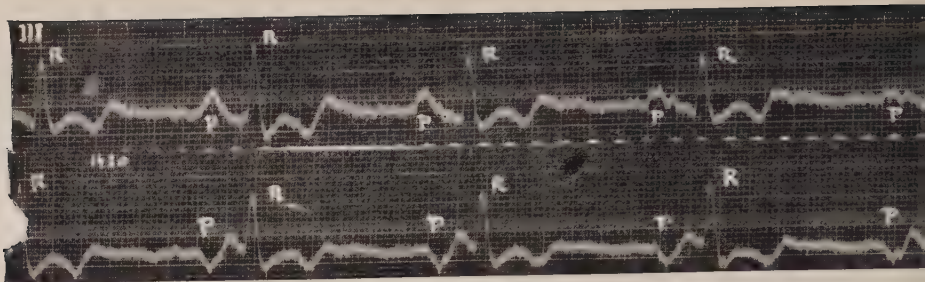


FIG. 151. Deformation of the auricular electrogram.

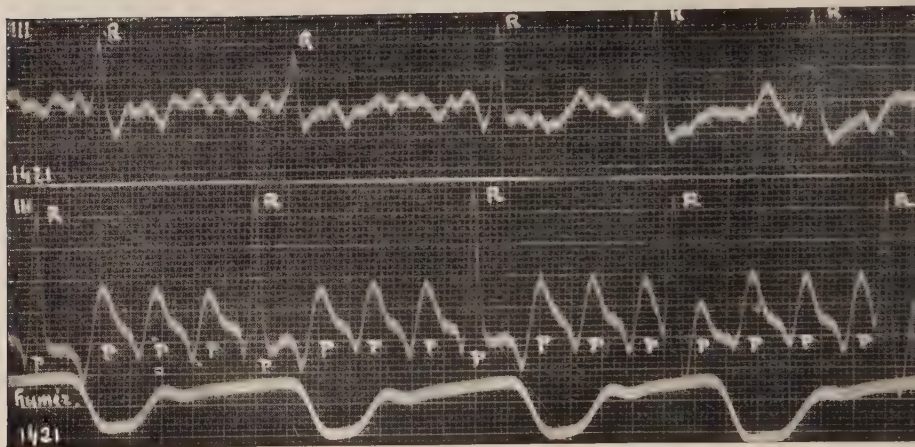


Fig. 152. Different appearance of two tracings of the same Lead III, taken from the same patient. The upper tracing is of the fibrillation type, the lower of the flutter type. These tracings and that in figure 151 are from the same patient.

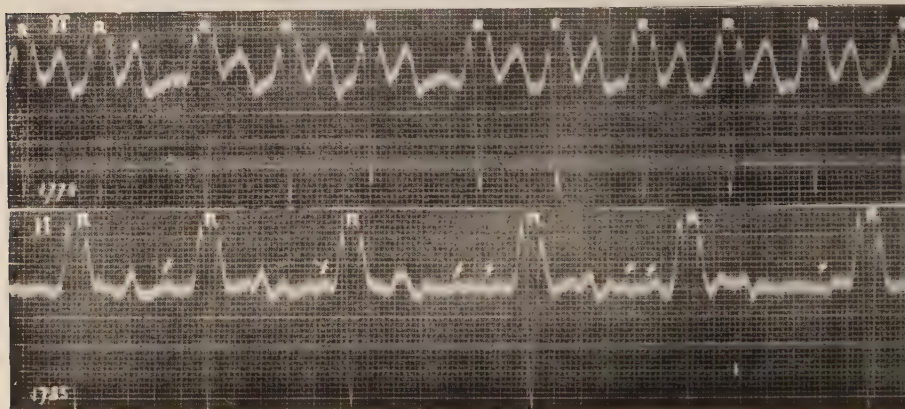
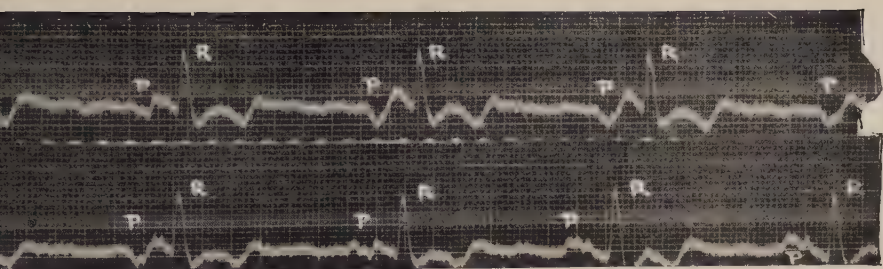
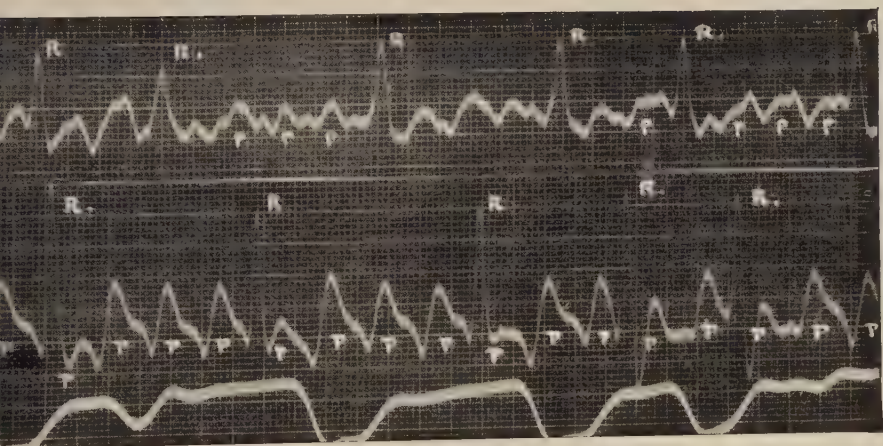


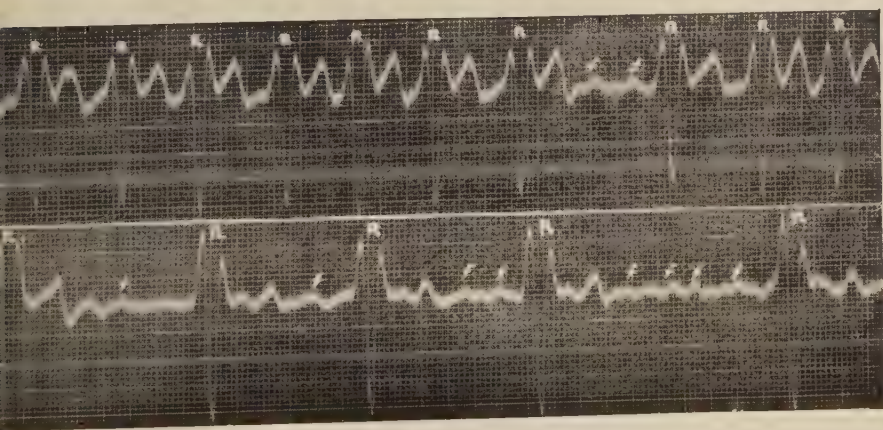
FIG. 153. An episode of ventricular tachyarrhythmia superposed on an auricular hypertachycardia. At the end of the tracing a ventricular pause occurred, unmasking the fundamental auricular rhythm. During the pause the ventricular rhythm was markedly slowed so that the fundamental auricular hypertachycardia was clearly visible.



the different aspects that P successively presents



at an interval of a few seconds, in a case of auricular tachyrhythmia. The upper tracing is the same patient. (The brachial curve is reversed, falling as the pressure rises.)



mia. The upper tracing, taken on January 14, 1927, shows a ventricular tachyrhythmia. The lower tracing was taken four days later. Under the action of dig-
tarythmia was disengaged.

and auricular electrograms is modified. We shall consider here only the auricular ones; in the upper tracing they give the aspect called that of fibrillation, and in the lower tracing that of so-called flutter. It is, however, a matter of the same anomaly of cardiac mechanism. But in the upper tracing the auricular electrograms have assumed a degraded form, analogous to that of the fourth and antepenultimate auricular beats of the tracing in figure 151, while in the lower tracing these auricular electrograms have taken the form analogous to that of the first P in figure 151.

I would remark that often in the auricular tachyrhythmias, Lead I gives a tracing very similar to that of fibrillation so called, where well formed P waves are lacking, while the P waves appear clearly in Leads II and III, and gives the tracing characteristic of flutter. Figures 118, 121 and 123 demonstrate this clearly.

Nevertheless that which sensibly differentiates flutter from fibrillation is the irregularity of the auricular rhythm in fibrillation. This irregularity exists at times in auricular tachyrhythmia of the flutter type, but is only slightly pronounced. On the other hand it is seen in fibrillation with more frequency. The degradation of the auricular complex renders it difficult to determine exactly the rhythm of the auricle and its regularity. Flutter and fibrillation are two closely related modes of the same anomaly of the cardiac mechanism, and should be classed and described in one and the same chapter on auricular tachyrhythmia and hypertachyrhythmia.¹

We may compare figures 148 and 138; the two tracings were obtained from the same subject, the first being of the hypertachyrhythmic type and the second of tachyrhythmic.

The ventricular rhythm is habitually irregular in auricular hypertachyrhythmia. It may be rapid, medium, or slow. At times its irregularity presents a certain regularity, as in flutter. The ventricular complexes may be grouped by twos, as in bigeminism (fig. 150), by threes, or by fours.

When a ventricular tachyrhythmia is joined to an auricular hypertachyrhythmia, the ventricular rhythm may be regular for quite a long time (upper tracing in figure 153). The ventricular rhythm may likewise be very regular in cases in which it is considerably retarded.

Ocular compression or the exhibition of digitalis may slow the ventricle; but the auricular tachyrhythmia will not be modified and will reappear between the lengthened ventricular complexes (lower tracing in figure 153).

Explanation of Auricular Tachyrhythmia

If auricular tachyrhythmia, or flutter, and hypertachyrhythmia, or fibrillation, represent two types of the same species which differ only in the velocity of the rhythm, the same explanation will hold good for both.

We believe that auricular tachyrhythmia originates always from the overactivity of the atrionector which rules the functioning of the auricle. This overactivity may result from nervous, humoral, or toxic factors, which we invoke in every case. When these factors act on the atrionector alone or more upon it than on the ventriculonector there will result a discordant auricular tachyrhythmia. If the factors act equally the ventricular rate will equal the auricular, as in the paroxysms which we have described.

The overactivity of the cardionectors, especially that of the atrionector, which brings about the auricular tachyrhythmia, seems to us to be more frequently closely associated with an increased blood circulation in this atrionector. The hyperemia may be active through increase of the arterial circulation, but it is probable that there is more frequently a passive hyperemia, due to some obstruction to the venous outflow.

The auricular acceleration may then in its turn increase the obstruction to the intra-auricular venous circulation and so increase the passive hyperemia not alone of the atrionector but of the ventriculonector as well. This, in turn, occasions a more rapid ventricular contraction which further increases the hyperemia and so contributes to the acceleration of rhythm. In this way is created a sort of vicious circle manifested by a crisis of double tachyrhythmia—auricular and ventricular. The paroxysm ceases only when the vicious circle is broken by the occurrence of a ventricular pause causing anemia of the cardionectors.

Ventricular arrhythmia, often observed in flutter and almost constantly in fibrillation, also seems to us to cause disturbances of the venous circulation of the cardionectors. These troubles increase with the acceleration of the auricular rate. The right auricle tends to become distended and its contractions are but little or not at all effective—all conditions eminently favorable to hinder the emptying of the veins of the cardionectors and to prolong unduly their contact with a modified fluid medium.

The condition is much the same in patients having a mitral lesion and consecutive dilatation of the right heart, especially of the right auricle, where we find with extreme frequency the anomaly of cardiac mechanism which we are here studying.

Arrhythmia of the ventricle disappears or is lessened under two conditions—either when the beat is too rapid or when it is too slow. This is, we believe, because in the first case the ventriculonector, already in a state of active arterial hyperemia, is less susceptible to the variations of venous hyperemia. In the second case the dominant element which influences the functioning of the ventriculonector is its arterial ischemia which tends to compensate the venous stasis.

The independence of the two cardionectors and their circulation accounts for all observable combinations, in which different modalities of functioning of the auricle and ventricle—acceleration, retardation, regularity, irregularity—are associated.

I would add a remark to the preceding exposition. It is possible that the auricle may fibrillate, that is to say that its wall may be the seat in man of parcellate or fibrillary contraction. This has been observed experimentally in animals when the heart is anemic or when the muscle is electrically excited. But the facts classed under the name of auricular fibrillation and which correspond to the tracings considered as characteristic of this anomaly of auricular functioning seem to me, in the absence of proof to the contrary, to originate from a total contraction of the auricular walls, more rapid and less regular than in flutter, but of identical nature; not from parcellate fibrillation. It seems to me that it is difficult to range in the same category the transitory phenomena observed in the experiment animal, phenomena which are often preagonic, and the permanent phenomena seen in man which may, barring accident, persist for years.

Other Hypotheses. Having explained our understanding of auricular tachycardia, whatever be its type—flutter or fibrillation—it is proper to present other theories regarding its origin and nature.

Flutter. Lewis maintains regarding flutter, the hypothesis of what is called “circus contraction” (Garrey²) or “circus movement” (Lewis). It rests on an experiment made on a muscular ring taken from a jelly fish (Mayer 1908³). If this muscular ring is irritated at a definite point, a wave of contraction starts to the right and left of the region affected. The two waves uniting at the opposite extremity of the ring annul each other and the contraction is arrested. But if a circumscribed part of the ring is lightly compressed by a forceps and a point, situated at the right, for example, of the region compressed is immediately stimulated, there is only one wave of contraction, that going to the right. The wave going to the left is blocked by the part compressed. If, then, the compression is loosened before the wave of contraction in its course around the ring has reached that region, the blockade is raised, and the wave of contraction passes the segment recently blockaded and extends beyond the point from which it started, the refractory period of which, following the first contraction, having just ceased. The wave of contraction always finding in front of it a portion of the ring which has just emerged from its refractory period, follows it indefinitely. In practice, as a consequence of a single stimulus, a wave of contraction has thus circulated for hours in the muscular ring.

An analogous result has been obtained with a ring from the heart of a

tortoise. From his experiments on the dog, Lewis has concluded that the course followed by the circus contraction surrounds the orifices of the vena cava superior, pulmonary veins, and vena cava inferior. From this circle radiating waves detach themselves to gain all the parts of the auricle. The circular wave and the radiating waves will always have the same course.

It results from this hypothesis that the flutter will be more likely to be established in proportion as the stimulation travels more slowly and the refractory period of the muscle is of shorter duration.

It may be objected to Lewis's theory of the "circus contraction" that it is difficult to understand the persistence of this circular course of the wave of contraction, for it is admitted that it may penetrate equally into other muscular territories. One cannot, then, comprehend how the circular wave alone finds expression in the electrocardiogram while the centrifugal radiating waves do not manifest themselves there.

De Boer⁴ has proposed another explanation of flutter. On account of the poor nutrition of the auricular myocardium all of its parts do not lose their refractoriness at the same time, and consequently a stimulus does not cause their contraction at the same time; the action is fractionated, not becoming effective until the refractory period is passed. Flutter then betrays a fractional contraction of the auricular myocardium. And this mechanism will be prolonged so long as the wave of excitation finds in front of it a non-refractory portion of the myocardium. De Boer holds that the stimulus proceeds from the sino-auricular node.

The anisorhythmia would result, according to the classical theories, from two causes. In every case the stimulus originating at the level of the auricle, whether normotopic or heterotopic, reaches the ventricle and causes its contraction. But the auricular stimulus is not always effective, either because it reaches the ventricle at times which do not coincide with the refractory phase of the latter or because the stimulus is blocked at certain periods in the bundle of His.

Fibrillation. It is admitted, since Hering, that the auricle in a case of fibrillation is in a special condition. There is incessant activity, not of the cavity, properly speaking, but of the wall. The essential condition for preventing the auricular contraction occurring as a whole, in a coördinate fashion, is a particular state of the myocardium which prevents the stimulation from passing through it with the same regularity and the same facility as normally.

The hypotheses differ as to whence proceeds the excitation. Three answers are made to this question, answers which we will briefly quote.

Theory of Monotopia. This theory, defended by Rothberger and Winter-

berg,⁵ assumes that the stimulation proceeds from a single center. This is not the sino-auricular node but a heterotopic center which emits a rapid series of excitations. The refractory phase of the auricular tissue would then be extremely short. It would result from these special conditions that the portion of the myocardium close to the center of excitation would cease to be refractory and again become irritable, while the portion of myocardium more remote from this center would be again in the phase of contraction. A new excitation would then act on the near portion while at the same time the preceding excitation was acting on the distant portion. Two or more waves of excitation, starting from the same center would follow one another then in their auricular course. The algebraic sum of the electrical phenomena would vary according to the proportion of the contracted to the relaxed portions.

Theory of Polytopia. This theory, first presented by Engelmann, accepted and then abandoned by Lewis, has again been defended by Kisch.⁶ A stimulus of a nature not specified originates at numerous points of a hyperexcitable myocardium and engenders contractions which, joining each other or interfering, give rise to the irregular events of the curve.

One objection among others to this theory is the fact of experience that a normal rhythm may abruptly follow the phase of fibrillation. The sino-auricular node would then have reassumed control. It is inexplicable that the accessory centers should all be arrested at the same instant.

Theory of "Circus Contraction." According to this theory, advanced by Lewis, auricular fibrillation differs from flutter in that the muscular ring traversed by the wave of excitation is smaller and more sinuous and so sends out sinuous centrifugal excitations. No positive fact is furnished to support this theory.

Theory of Fractional Contraction. De Boer has proposed this theory, according to which the excitation is supplied by a single center, which may be the sino-auricular node. The condition necessary and sufficient for the production of auricular fibrillation is the state of the auricular muscle. De Boer rests his theory on the following facts: A galvanic shock on the heart of a frog, exsanguined for some time, produces fibrillation if the shock follows immediately the end of a systole; it results in an extrasystole if it is a little deferred. De Boer concludes from this that when the myocardium is well nourished, its phase of contraction occurs everywhere at the same time, and it is the same with the refractory phase. When, therefore, the excitation supervenes at the end of this general phase, there is a general contraction of the auricle; but when the myocardium is poorly nourished its different portions are not all in the same phase at a given moment.

Some parts are in contraction, others in the refractory phase, others again in a non-refractory phase. When an excitation is launched through this mosaic of myocardial territories in different phases, its course will be irregular and it will cause a series of partial contractions which succeed one another in a jerky fashion, whence the tracing of fibrillation. As long as this differentiation of the myocardium into districts with different phases persists, the excitation will continue to traverse the auricle in this jerky fashion. De Boer extends this theory to the ventricle as well, and explains ventricular fibrillation in the same way.

In flutter as in fibrillation the ventricle will contract when an auricular stimulus succeeds in passing through the bundle of His. In flutter, where one auricular beat out of one, two, or three is followed by a ventricular beat, it is, they say, because one of the times the stimulus passes, while at the others it is blocked. In fibrillation it is still the auricular stimulus which sets off the ventricular contraction, but there must be an actual filtration through the bundle of His of the multiple excitations which furrow the auricle. The bundle of His opposes their transmission as a whole and blocks them in part. An interruption by the bundle might block all the auricular stimuli, as shown in a case reported by L. Frédéricq; the ventricle then beats by itself, more slowly and almost regularly.

ANISORHYTHMIA DUE TO DISCORDANT VENTRICULAR TACHYRHYTHMIA

Simple Ventricular Tachyrhythmia

A good type of anisorhythmia, due to ventricular tachyrhythmia, is, I think, that represented by volleys of ventricular proiosystoles bursting in on an auricular rhythm of medium rate. But, of course, it is then necessary to accept my view that there is no essential difference between a normal ventricular systole and a ventricular proiosystole, as both depend on the functioning of the ventriculonector. If, on the other hand, one adhered to the classical theory which attributes the proiosystole (extrasystole) to a stimulus originating in an ectopic center, it would be necessary to drop most of the cases from this chapter, for authentic cases of anisorhythmia due to ventricular tachyrhythmia would then be very rare. When there is ventricular tachyrhythmia the events are in fact so crowded together that it is often impossible to count the P waves swamped in the R or T waves of the tracing.

It is nevertheless possible to prove by the electrocardiograph the existence of anisorhythmia due to ventricular tachyrhythmia. I have borrowed from Pardee a typical curve of this kind in which the rate of the auricular

contraction is 103, while that of the ventricular beat is 180 per minute (fig. 154). The author, however, interprets this tracing quite differently. He considers it as a curve of nodal tachycardia combined with complete atrioventricular dissociation.

Dr. Gallavardin⁷ has published a remarkable example of this anomaly, the rhythms having the relations: 94 to 108, 100 to 193, and 94 to 194.

Ventricular Hypertachyrhythmia (Ventricular Fibrillation)

It is admitted that the ventricle, like the auricle, may fibrillate, that is to say, may be the seat of fibrillary contractions, non-synergetic and ineffec-

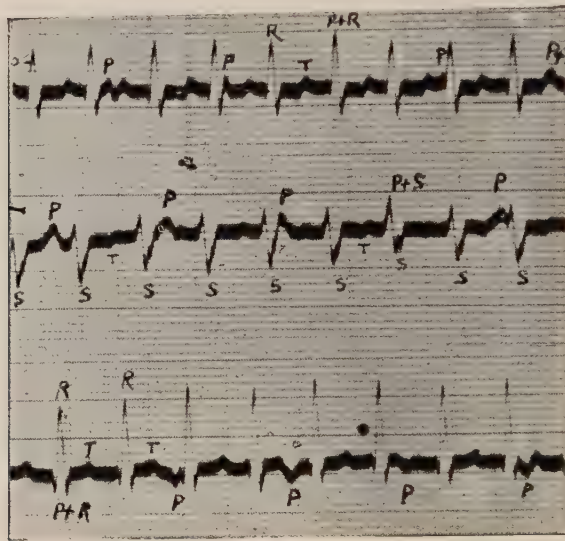


FIG. 154. Anisorhythmia due to discordant ventricular tachyrhythmia. (Reproduction of a tracing by Pardee.) The ratio of the beats is 103:180.

tual. Ventricular fibrillation, unlike auricular, would be rapidly fatal. That fibrillation is seen in the auricle and ventricle is a fact of observation established in the case of the animal heart and one which cannot be called into question. But it is by no means proven that experimental auricular fibrillation corresponds to auricular tachyrhythmia, improperly termed complete arhythmia. Neither have we any authority for believing that ventricular fibrillation is seen clinically in man. The tracings which have been given in support of this hypothesis do not carry conviction. These tracings are of two kinds. In the first type of curve we recognize easily

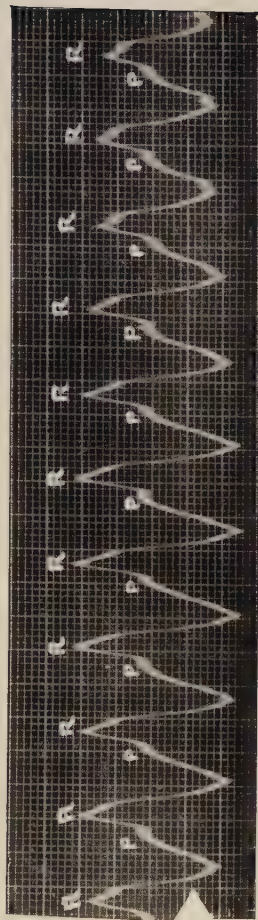
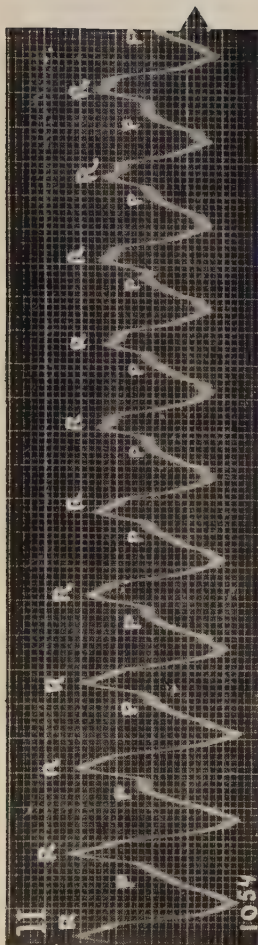


FIG. 155. An episode of ventricular tachyarrhythmia superposed on an auricular tachyarrhythmia. There is a false appearance of "ventricular fibrillation." This tracing is from the same patient as the one from whom the tracing in figure 115 was taken. The ratio of the rhythms is 250:250.

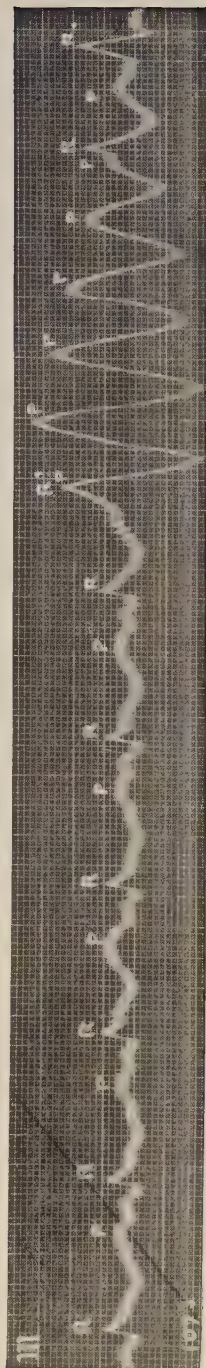


FIG. 156. A brief attack of auricular tachyarrhythmia, giving the false appearance of "ventricular fibrillation"

the ventricular complexes; they are evidently deformed and may follow one another irregularly, but nevertheless they reveal a total contraction of the ventricular cavity and not fibrillary contractions of the wall. I would instance as examples of this type figures 300 and 302 given by Lewis in the second edition of his treatise, and figure 12 of Clerc⁸ reproduced in an article by Kerr and Bender. In the latter a ventricular tachyrythmia seems to be combined with auricular hypertachyrythmia.

A second type is characterized by a tracing like the movements of a violin bow which is interpreted as recording the ventricular contractions. It is shown in a tracing by Hoffmann in figure I of plate II; in one of A Weber (fig. 129); in tracings of Lewis (figs. 297, 299, 301 of the second edition of his book), and again in one published by Gallavardin and Berard.⁹ All of these "fiddle-bow strokes" seem to me to be related to the auricular movements. They resemble almost exactly the tracing in fig. 155 which belongs to the patient from whom was obtained the tracing in figure 115. I interpret the two tracings as two short paroxysms of auricular tachyrythmia interrupting a normal rhythm (figs. 156, 157). This interpretation of the second of these tracings may, however, be objected to. Certain sharp summits capping the sinusoidal wave at a distance from R just about equal to the interval PR at the beginning and end of the tracing, may be considered as P waves. To what then can we attribute the waves which we have already connected with the auricular systoles? We have observed this anomaly several times, but have no satisfactory explanation to give of it.

ANISORHYTHMIA DUE TO DISCORDANT AURICULAR BRADYRHYTHMIA

Cases of discordant auricular bradyrythmia are rare. I have borrowed from Schrumpf and from Lewis two tracings which are of this type. I have reproduced the tracing of Schrumpf on the same plan as all the other tracings in this book, so that it may be compared with them (fig. 158). The author does not give any details of the case but interprets the curve as caused by a block in the bundle of His, complicated by a sino-auricular block, which simply means adding one hypothesis to another. I should be inclined to regard the curve as showing the effect of a hypoactivity of the two cardioneotors, more marked in the atrioneotor. A stenosing lesion of the arteries of the cardioneotors, the obstruction being greater in the artery of the atrioneotor, would be consistent with the ascertained facts.

Lewis has published a curve of the same type which is also reproduced here, redrawn to scale (fig. 159). It is a tracing of an experiment on a cat, asphyxiated in two stages and presenting an auricular and ventricular bradyrythmia, with predominance of the latter. The auricular brady-

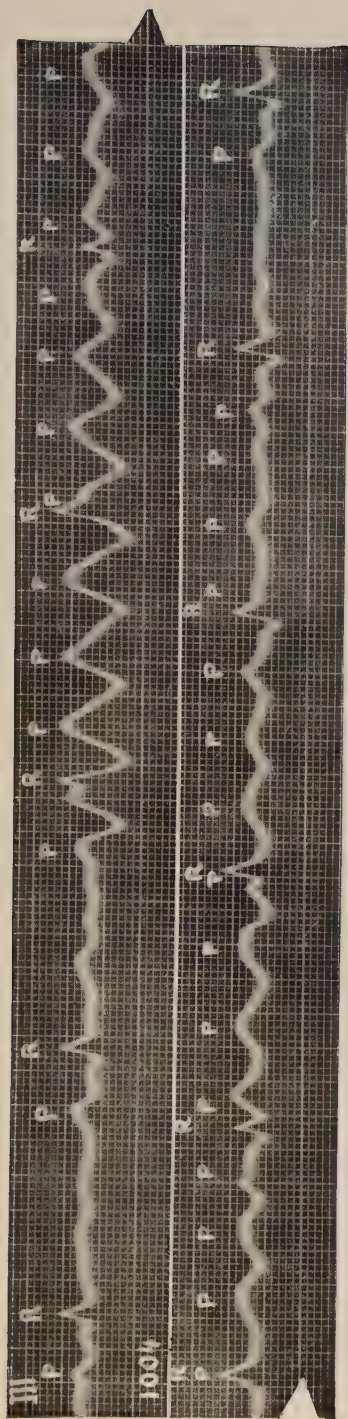


FIG. 157. A brief attack of auricular tachyrrhythmia, giving the false appearance of "ventricular fibrillation"



FIG. 158. Anisrhythmia due to discordant auricular bradyrrhythmia (Schrumpf). The ratio of the rhythms is 20:37



FIG. 159. Anisrhythmia due to discordant auricular bradyrrhythmia (Lewis). The ratio of the rhythms is 23:48

rhythmia became predominant in its turn, after refrigeration by the application of ice over the region of the atrionector. We see here a diminution of the activity of the two cardionectors, predominant at first in the ventriculonector, later in the atrionector. A diminution of the arterial supply locally, by refrigeration of the atrionector, renders a probable explanation of the phenomenon.

ANISORHYTHMIA DUE TO DISCORDANT VENTRICULAR BRADYRHYTHMIA

The curve in figure 160 is an example of this anomaly. The P waves follow one another at the rate of 120, the R waves at a rate of 40. The auricular rhythm being by convention more frequently taken as basis of comparison, there is relative ventricular bradyrhythmia.

The Auricular Complexes

Certain among these, as in auricular tachyrhythmia, are no longer covered by the ventricular complexes and so we may obtain an idea of their general form, P is only the initial event of the auricular complex. We see on the tracing a depression preceding P, the diphasic aspect of P and the double wave which, with Hering, we call Ta. This appearance is particularly evident on this tracing; as a rule it is less marked.

The rhythm of the auricular complexes may be regular or irregular, regularity being the rule. But even in this case the interval between two P waves is not rigorously constant and in particular it is to be noted that on each occasion a ventricular complex is inserted between two P waves. We have already spoken of this fact which we interpret as a premature functioning of the atrionector, under the influence of the wave of blood propelled into the arteries by the ventricular systole (fig. 161).

The Ventricular Complexes

The form of the ventricular complex is most frequently normal, but it may be distorted. Its duration is inversely proportional to the velocity of the rhythm. In figure 162 we see two ventricular complexes occurring in the same subject with an interval of a few minutes. The length of the complex is 34/100 second when the rhythm is 63, and 44/100 second when it is 36 per minute.

The ventricular rhythm may be regular or irregular. When it is regular and the auricular rhythm is also regular, we are justified in speaking of a relation between the two.



FIG. 160. Anisorhythmia due to discordant ventricular bradyrhythmia. "Partial block" type. Ratio of the rhythms, 120:40—3:1



FIG. 161. Anisorhythmia due to discordant ventricular bradyrhythmia. The ratio of the rhythms is 78:40. Note that the intervals between P_1 and P_2 , P_3 and P_4 , P_5 and P_6 , in which an R wave is interpolated, are shorter than the intervals between P_2 and P_3 , P_4 and P_5 , P_6 and P_7 . "Total block" type.

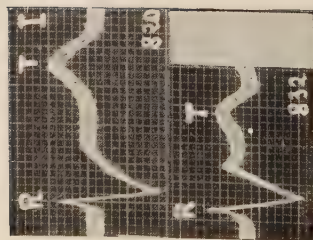


FIG. 162

FIG. 163

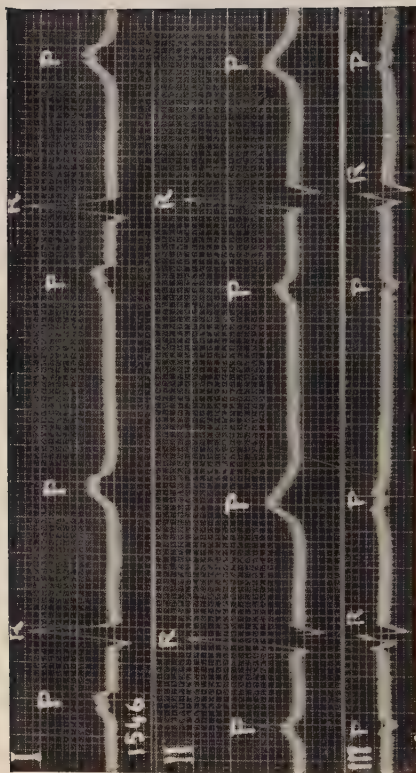


FIG. 162. Comparative length of the ventricular complex in a slow or normal rhythm. The upper complex measures 44/100 second and corresponds to a rate of 36. The lower complex measures 34/100 second corresponding to a rate of 63. (Tracing retouched.)

FIG. 163. The engulfing of P in wave T. This gives rise to the danger of failing to recognize the anisorhythmia due to discordant ventricular bradyrhythmia of the ratio 100:50. P being reversed in Lead III, distinctly notches the apex of T. This is foreshadowed in Lead I. If Lead II only had been registered, there would have been great risk of failing to recognize this. Nevertheless the interval PR, which measures 16/100 second, ought to have aroused suspicion.

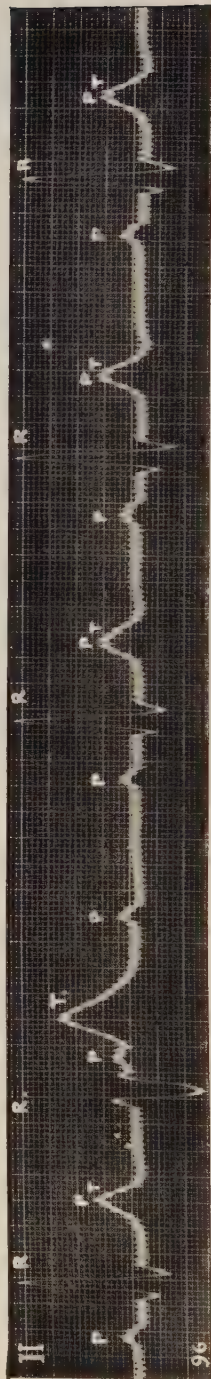


FIG. 164. Engulfing of P in wave T. This would have been in danger of not being recognized if the protosystole, breaking the ventricular series, had not momentarily unmasked the auricular rhythm and shown that there was anisorhythmia from discordant ventricular bradyrhythmia with the ratio 112:56.

Interrelation of the Auricular and Ventricular Rhythms

This relation may be simple. It is so when the quotient of the number of auricular pulsations divided by the number of ventricular pulsations during the same length of time is a whole number. It results from this simple relation that the auricular complexes seem to constitute a fixed group with certain ventricular complexes.

The tracing in figure 163 corresponds to the ratio $\frac{100}{50} = \frac{2}{1} = 2$. Once in two times P is followed by R. The type $\frac{2}{1}$ leads at times to confusion. The tracing in Fig. 163 on hasty inspection may appear to be a tracing of a rhythm rather slow (50), but without anything unusual save the length of PR, which is 26/100 seconds. The T wave is midway between two quite clear P waves, which frame it. But there is one P wave engulfed in this T. It is easy to assure oneself of this by carefully comparing the tracings in the three leads. In L. III, in fact, where P is negative, we see that the peak of the T wave is notched and the wave is really P plus T. In L. II where P is positive, its peak is added to that of T, so that P passes unrecognized. In L. I the notched aspect of P is found again in the peak P plus T. But if one saw only Lead I, one would probably have failed to recognize the submergence of P in T. Sometimes a sudden breaking of the ventricular rhythm by proisystoles reveals the true auricular rhythm, as in the tracing given in figure 164.

The tracing in figure 160 shows a relation $\frac{120}{40} = \frac{3}{1} = 3$. That in figure 165 shows a relation of $\frac{100}{25} = \frac{4}{1} = 4$.

The tracing in figure 166 presents in its first half the ratio $\frac{66}{66} = \frac{1}{1} = 1$. The ratio $\frac{1}{1}$ in such a case gives a tracing quite similar to a normal one. If one had only the evidence of this tracing one would certainly fail to recognize the fundamental disturbance of the cardiac mechanism. Sometimes, however, a certain peculiarity raises a doubt. This may be for example, as we have just now seen, the abnormal length of the PR interval which may be 22 to 24 hundredths of a second. But this elongation does not always exist and in fact it is lacking in figure 166, where PR measures 16/100 second. In this limited case there is no longer any ventricular bradyrhythmia.

In the same manner in auricular tachyrhythmia, where the rate of the ventricle increases until it equals the auricular rate, we have the same type,

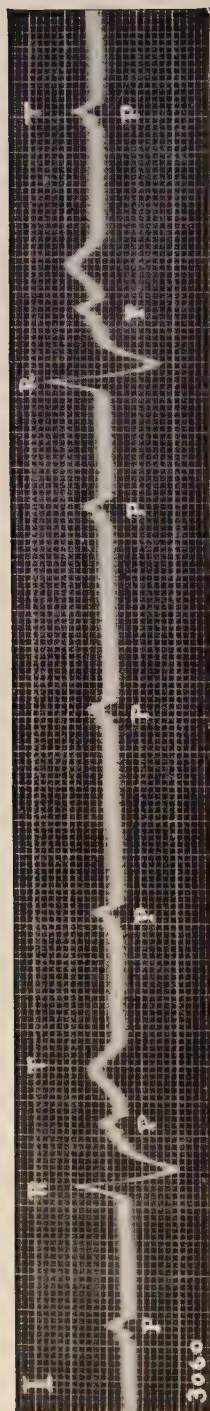


Fig. 165. Anisorhythmia from discordant ventricular bradyrhythmia. The ratio of the beats is 68:17 - 4:1. So-called "partial block" type. Note the considerable length of PR, which measures 56/100 second, and compare this with tracing 176, another case of partial block where PR measures 56/100 second.

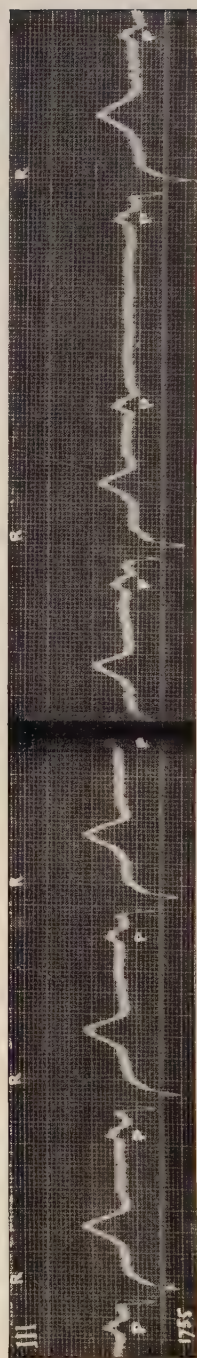


Fig. 166. Spontaneous variation of the ventricular rhythm, determining now isorhythmia and again anisorhythmia. The two halves of this same film were taken at an interval of several seconds and show clearly the change in the ventricular rate passing from 66 to 33, while the auricular rate does not change. When the ventricular rate equals the auricular the tracing appears perfectly normal. The interval PR is 14/100 second, i.e., an apparently isorhythmic type. A slight change in the ventricular rate reveals the anisorhythmia from discordant ventricular bradyrhythmia. This passage from the anisorhythmia to the anisorhythmic type occurs frequently in this patient. It should be compared with the analogous phenomenon that we saw in the chapter on anisorhythmia due to discordant auricular tachyrrhythmia in which the ventricular rhythm sometimes becomes as rapid as that of the auricle. It will be noted, in the second half of the tracing that the interval between two consecutive P waves diminishes when an R wave is inserted.

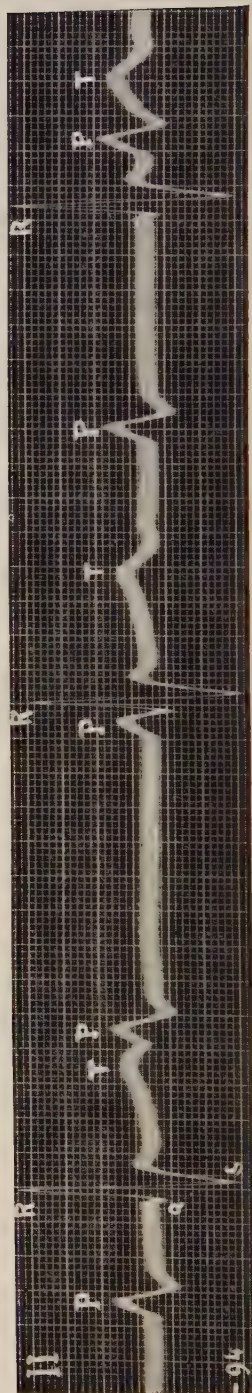


Fig. 167. Anisorhythmia due to discordant ventricular bradyrhythmia. The so-called "total block" type. The ratio of the rates of the

$\frac{1}{1}$; there is no longer, properly speaking, auricular tachyrhythmia. Facts break down all our classifications. The auricular rhythm remaining fixed, the ventricular rhythm may change, but the ratio of the rhythms may remain a whole number. The tracing in figure 166 gives in its first part a relation $\frac{66}{66} = \frac{1}{1} = 1$; in the second part, taken immediately after, we have the ratio $\frac{66}{33} = \frac{2}{1} = 2$. In this case the change was spontaneous, but we may obtain it experimentally by compression of the eyeball which acts on the ventricle and not on the auricle.

In all these tracings, following the ratio of the rhythms, on condition that this ratio shall be a whole number, P is followed by R every time, or once in two, three, or four times. This particular arrangement is termed "partial block"; later we shall explain why.

When the ratio of the rhythms, both remaining regular, is no longer a whole number, we have a different arrangement. The tracing in figure 167 shows that the P and R waves may assume any position in relation to each other, there seeming to be no relation between them.

In reality the auricular and ventricular complexes behave like the division on the two scales of a vernier of which the zeros were once for all dissociated by an interval PR. The divisions would be thrown into discordance, but after a certain distance they will have come back to about the same relation as at first. The interval before the return of the two to their original arrangement will depend on the numerical relation between the two unequal rhythms. One may thus encounter types corresponding to the ratios 3:2, 4:3, 5:4, or 6:5. After three PP intervals we find again the initial grouping PR, while two RR intervals will be evolved during the same time, whence the ratio of 3:2—and the same with the other ratios. If the period is too long it overlaps the usual length of the film, and so it fails to be recorded in its totality with all its dimensions and therefore is not recognized. The tracings in which the ratios of the rhythms are not whole numbers, and where as a result the P and R waves seem to have lost all group relation, are denominated classically as "total block."

The rhythm of the auricle remaining regular, the functioning of the ventricle may be irregular, but this irregularity may nevertheless affect a certain regularity, the ventricular systoles being regularly grouped by twos, threes, or fours. This grouping may be repeated every time, giving the appearance of bigeminism, as in figure 168. Of the two approximated, ventricular complexes, the first corresponds to a systole, the second to a

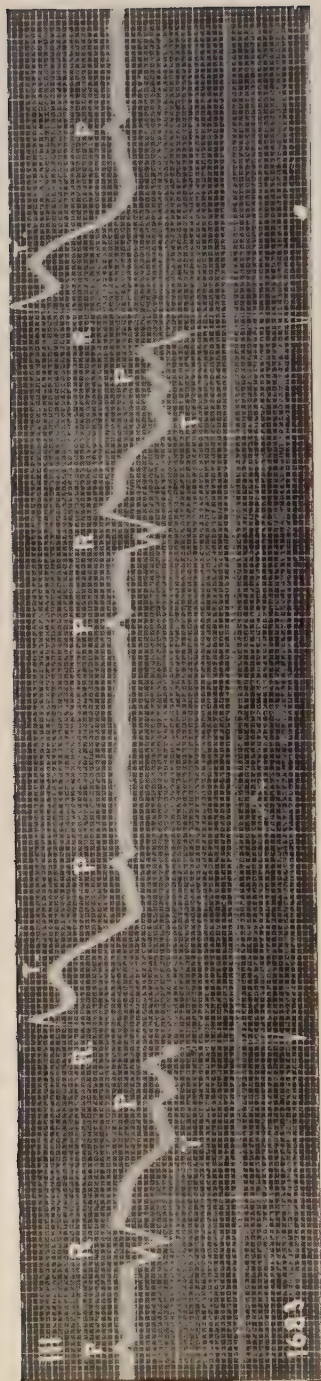


FIG. 168. Anisorhythmia from discordant ventricular bradyrhythmia. Ventricular allorhythmia by bigeminism

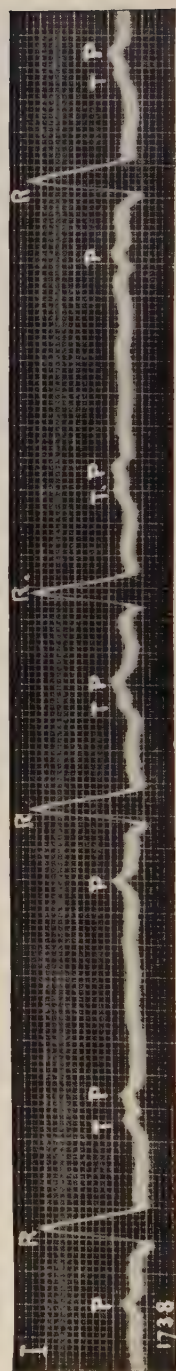


FIG. 169. Anisorhythmia from discordant ventricular bradyrhythmia. Rupture of the ventricular series by a proiosystole



FIG. 170. Anisorhythmia from discordant ventricular bradyrhythmia. Rupture of the ventricular series by a group of one systole and two protosystoles. This tracing and that in figure 168 were furnished by the same patient.

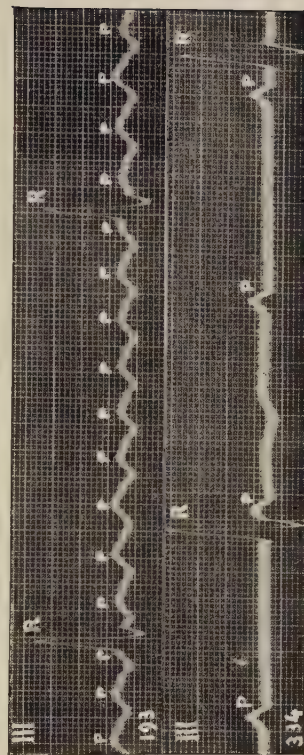


FIG. 171

FIG. 171. An episode of auricular tachyrhythmia superposed on a ventricular bradyrhythmia. The ratio of the two beats is $326:37 = 9.08$.

FIG. 172. Anisorhythmia from discordant ventricular bradyrhythmia. This tracing and the preceding one were taken from the same patient at an interval of five months.

In the second tracing the auricular tachyrhythmia has disappeared, but the anisorhythmia from ventricular bradyrhythmia persists, the ratio being $78:33 = 2.03$.

FIG. 172

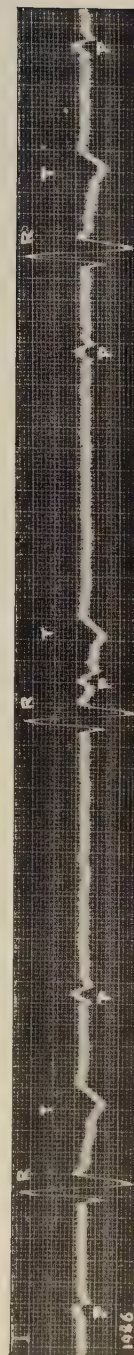


FIG. 173. Anisorhythmia from preponderant ventricular bradyrhythmia. There is here a conjoint bradyrhythmia, auricular and ventricular; but the ventricular rate, much slower than the auricular, determines the anisorhythmia. The ratio is $40:27 = 1.48$.

proiosystole, the complex of which is deformed. This is quite frequent when digitalis has been given in large doses and for a considerable period. But the proiosystole may also be in a well formed complex (fig. 169). Figure 170 shows three systoles grouped regularly, of which the two last of each group are of a deformed complex with difference in deformity. The functioning of the ventricle may be quite irregular.

We have up to this point considered only the cases in which the count of the auricular rhythm has not exceeded mean values. But the auricular rhythm may, while remaining regular, assume values differing from the normal; whence the possibility of new combination between the two rhythms.

The auricular rhythm may in fact be notably faster or slower than the average. It may be more rapid, and we find then a ventricular bradyrhythmia together with an auricular tachyrhythmia. Figure 171 shows this combination with the ratio $\frac{336}{37} = 9.08$. The tracing shown in figure 172 was taken from the same subject five months later and presents a ratio of $\frac{87}{43} = 2.03$.

It may be slower than the average so that it is only by convention that we speak of ventricular bradyrhythmia. There is a coupled auricular and ventricular bradyrhythmia, the ventricular being relative. Such facts are quite rare. I give a good example of them below, where the ratio of the rhythms is $\frac{40}{27} = 1.04$ (see fig. 173).

The auricular slowing may be such that the auricular and the ventricular rates are equal. Nevertheless it will be suspected that this is not a case of simple slowing of the cardiac mechanism if several tracings from the same subject, taken at different times, are compared. We may also try to alter the rapidity of the rhythms, while taking the same tracing, by compression either of the eyeballs or of the pneumogastric in the neck, or by an inhalation of nitrite of amyl. The auricular and ventricular rates are then unequally modified and the anomaly of the cardiac mechanism, the anisorhythmic basis, is revealed.

Certain tracings of ventricular bradyrhythmia present an exceptional aspect, and there is no trace of an auricular complex. This is represented in figure 174, the three leads being shown. There are three hypotheses to account for this, the first of the following being the most probable. (1) There is a masked auricular tachyrhythmia; (2) there is a submerging of the auricular into the ventricular complex; (3) there is no auricular complex. I have not been able to see the patient again from whom this tracing was

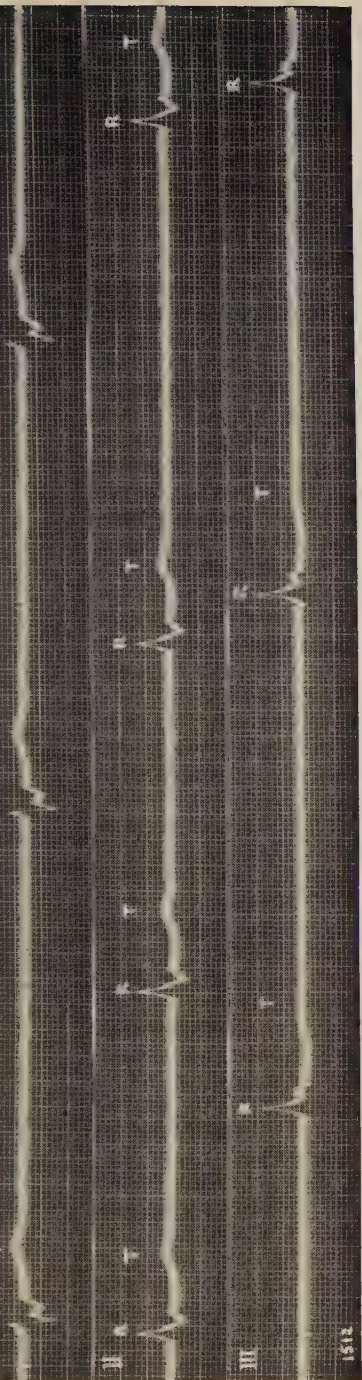


FIG. 174. A combination of auricular hypertachyrrhythmia and ventricular bradyrrhythmia. The auricular hypertachyrrhythmia is probable but does not show itself in the tracing. A registration in a precordial lead, as in that employed for the lower tracing in figure 150 would most likely have revealed a hypertachyrrhythmia of fibrillation type.

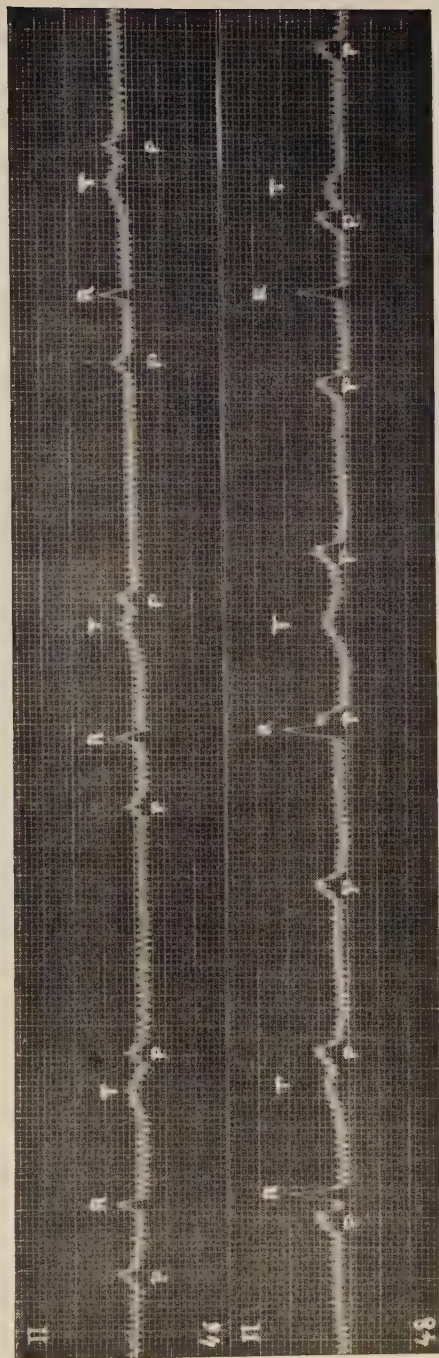


FIG. 175. Transition from the so-called "partial block" type to a "total block" type. The two tracings were taken at an interval of several minutes. In the upper tracing the ratio of the beats is $78:39 = 2:1 = 2$; there is a partial block. In the lower tracing the ratio is $104:39 = 2.66$; there is a total block. An acceleration of the auricular beat has sufficed to extinguish any apparent grouping between P and R, which is still tenable in the upper tracing.

taken, and to record the precordial leads which sometimes bring out the auricular complexes which are not revealed in the usual leads.

Anisorhythmia from discordant ventricular bradyrhythmia is explained classically as follows. The phenomenon called "partial block" (fig. 160) results from a default in the transit of the sinusal stimulus through the bundle of His. The conductivity of the bundle is then intermittent. When it fails temporarily all the stimuli leaving the sino-auricular node will be arrested, as is a railway train before the red light of the block system. There would therefore be a partial block. At the beginning of the Stokes-Adams syndrome, characterized by the phenomena of discordant ventricular bradyrhythmia which we are studying, the lengthening of the PR interval, which is here seen, betrays at times a diminished conductivity of this bundle. The usual theory seems therefore to be the most probable.

The phenomenon called "total block" (fig. 166) would depend on the suppression of the conductivity of the bundle of His. All the excitations are always arrested. The persistence of the ventricular contractions is explained by the action of the idioventricular center which would then function at a slow rhythm.

We see that according to this theory partial block and total block differ distinctly in their production. In the former a single center functions and the conductivity of the bundle of His is intermittent. In the latter two centers function and the conductivity of the bundle is nil.

Our interpretation is quite different. There is no essential difference between the phenomena, whether the block is partial or total. The auricular and ventricular complexes constitute two independent series which behave like the two scales of a vernier, with the understanding that the zeroes do not coincide but are separated by the length of the PR interval. According to whether the ratio of the auricular to the ventricular pauses is or is not a whole number, the complexes of one series will be disposed in a different way from those of the other, and will have the appearance of a partial or total block. The slightest change in the rapidity or regularity of one of the rhythms alters the ratio. Represented at first by a whole number, whence the aspect of the partial block, it is represented later by a fractional number whence the aspect of the total block. Figure 175 is an example of the passage from a partial to a total block. In the upper tracing the ventricular rhythm is 39, the auricular 78. The relation is $\frac{78}{39} = \frac{2}{1} = 2$; there is partial block. A few seconds later, as the result of the acceleration of the auricular rhythm alone, the relation becomes $\frac{104}{39} = 2.64$; there is total block.

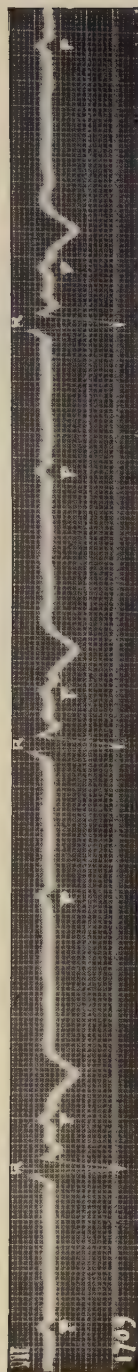


FIG. 176. Partial block in which the considerable length of PR excludes the hypothesis of any association between the auricle and the ventricle. The interval PR measures 56/100 second. The ratio of the beats is $2:1 = 2$. Compare the tracing in figure 165 in which the PR interval measures 35/100 second.



FIG. 177. Paroxysmal ventricular bradycardia. (The tracing was taken in an interval between the crises)

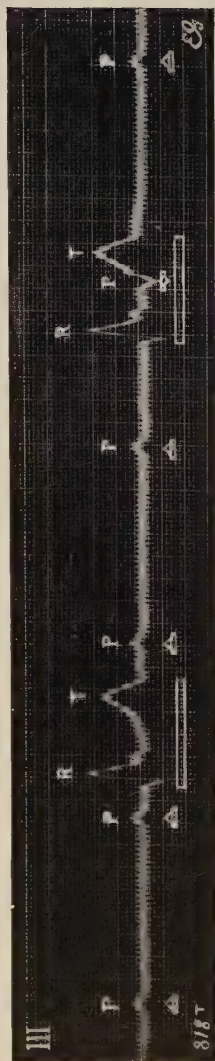


FIG. 178. Paroxysmal ventricular bradycardia, Tracing taken during a crisis

Certain tracings of a partial block may further have such an appearance that they exclude all idea of a direct relation between P and R (see fig. 176). The events always have the same relation to each other, for the ratio of the rhythms is a whole number, $\frac{72}{36} = \frac{2}{1} = 2$. But this position is such that the P wave nearest to the R is distant from it by 56/100 second, a duration which would be surprising to a partisan of the classical theory.

All of these modalities, partial block or total block, are simply explained if one grants that auricular and ventricular rhythms are, as always, independent rhythms. They may most frequently be unequal, a difference of activity in the functioning of the cardioneurons breaking the habitual equilibrium. More rarely, as in the preceding case, they may remain equal.

In auricular tachyrhythmia there is hyperactivity of the atrionector and in ventricular bradyrhythmia there is hypoactivity of the ventriculonector. The activity of the other cardioneuron remaining normal each time, each time there will be discordance of two series, auricular and ventricular, that is to say, anisorhythmia. There is no dissociation for there is no association by direct bond, the two rhythms being always independent. But sometimes, as in the normal heart, there is isorhythmia; sometimes as in the abnormal heart, anisorhythmia.

Hypoactivity of the ventriculonector, which brings about slowing of the ventricle probably arises from various factors. But it seems to us in the immense majority of cases to be due to ischemia. To this same ischemia we refer the ventricular hysterosystole which, coupled with a normal auricular systole, gives a lengthened PR interval. I would remind the reader that I have given in one of the preceding chapters (see p. 170) an anatomo-clinical observation proving an analogous relation between permanent auricular hysterosystole and a very tight stenosis of the artery of the atrionector. Ventricular hysterosystole is frequently, but not constantly, encountered in subjects who will later show a discordance of rhythms due to ventricular bradyrhythmia (Stokes-Adams syndrome), or perhaps between the crises of this bradyrhythmia. There is here only a question of degree. The ischemia induces hysterosystole or bradyrhythmia, according as it is more or less pronounced. Ischemia itself is due either to a tonic contraction or spasm of the musculature of the artery of the ventriculonector (transitory bradyrhythmia), or to a permanent narrowing of the lumen of the artery due to endarteritis (permanent bradyrhythmia).

ILLUSTRATIVE CASES

I will cite here the histories of three anatomic-clinical cases proving the coexistence of a stenosis of the artery of the ventriculonector and a ventricular bradyrhythmia.

Case I. Antoine D., aged 57, entered the service of Professor Vaquez at the Hôpital de la Pitié, June 24, 1925. During the preceding month he had had three syncopal attacks, in the course of one of which his pulse was 22 per minute. He was a vigorous man in whose previous history no disease was found, save a mild diabetes, recognized in 1923. Syphilis was denied and repeated Bordet-Wassermann tests proved negative. On admission there were seen numerous ecchymoses in the orbital and frontal regions and also on the limbs, these being due to a fall the previous evening. He complained only of general fatigue, and occasional spells of vertigo, occurring during repose, and even in bed, and independently of the syncopal attacks from which he feared a fatal outcome.

Since admission the patient had been in a bad condition, having suffered several syncopal attacks, even when lying in bed. The frequency of these crises diminished somewhat under the action of one milligram of atropine sulphate, administered during the course of 5 days.

The onset of the attacks was signalled by a sudden pallor; the eyes became dull, and loss of consciousness rapidly followed. Convulsive movements of the limbs occurred, lasting for four, five or occasionally twenty minutes, and ceasing as suddenly as they began. Before the attack the patient complained that his sight was blurred; he felt dizzy, and there was a sharp retrosternal pain radiating to the neck and head. During the attack the pulse, which was usually 66 per minute, fell to 32, 26, or 22.

Clinical examination in the intervals between the syncopal attacks gave little information. There was a slight metallic murmur in the area at the base of the heart, the sounds were muffled at the apex. The orthodiagram showed an organ of normal volume, the aorta, however, showed an increased area of shadow. Systolic blood pressure was 16.7 cm. (167 mm.). The lungs were normal.

The liver was slightly enlarged and there was marked tenderness in the epigastrium. A careful examination revealed no abnormality of the nervous system. There was polyuria, with glycosuria (excretion of 6 grams in 24 hours). Glycemia, to the extent of 1.18 grams per 100 was present.

On the 18th day after admission to the hospital the patient's condition became worse. All his previous symptoms returned. This condition lasted all night and on the following day he succumbed in an attack of syncope with slowing of the pulse to 10 per minute.

During his stay in the hospital, thanks to the installation of electrocardiography at a distance, given to the service by Professor Vaquez, we were able to take tracings while the patient was in bed, and as easily during the paroxysms as during the intervals.

On the tracing taken in the interval of the paroxysms (fig. 177) the most noticeable event was the marked elongation of the interval PR. Measured from the beginning of P to the beginning of R, PR was equal to 23/100 second. QRST measured 38/100 second.

The tracing taken in the course of one of the paroxysms of bradysphygmia (Fig. 178) showed a very distinct difference between the auricular and ventricular rhythms. The P waves succeeded one another at sensibly equal intervals. The usual rate of 66 to the minute rose to 73, but the ventricular rate dropped from 66 to 30. There is no simple relation between the numbers 73 and 30, so the discordance was complete. QRST measured 48/100 second.

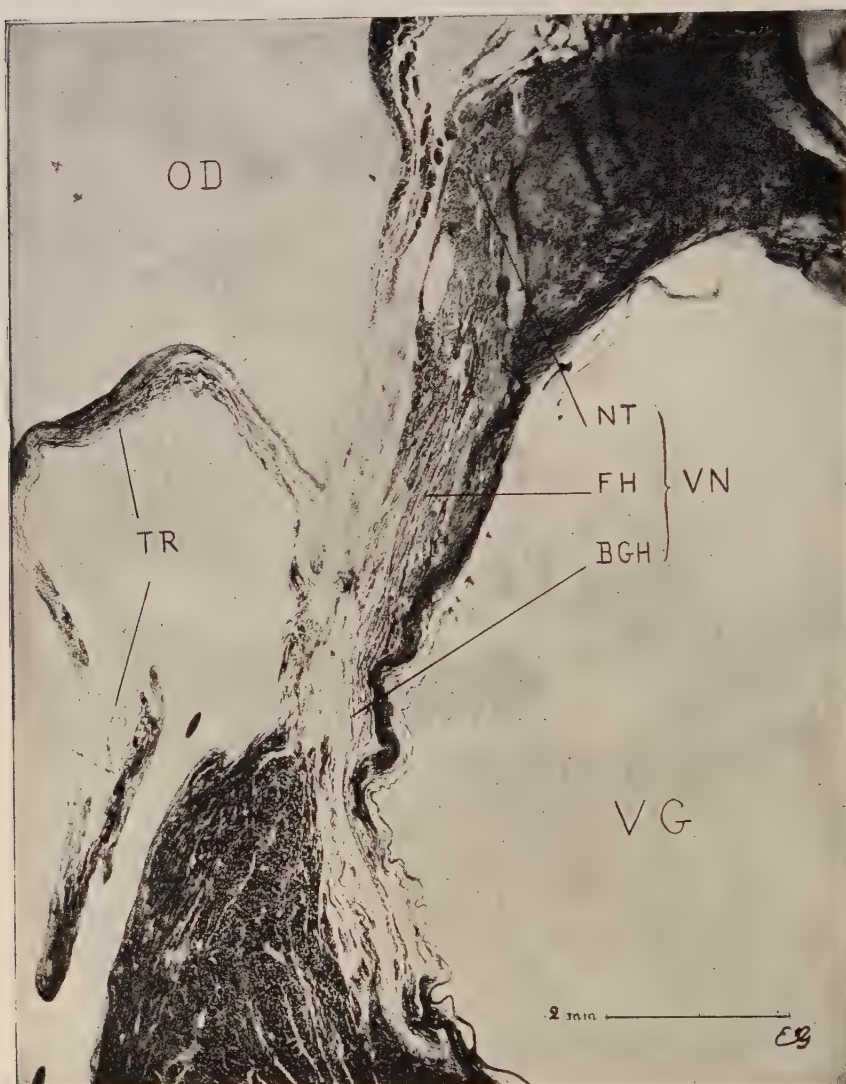


FIG. 179. The entire ventriculonector. Microphotograph of section 434, showing the detail of structure of the ventriculonector. VN, plexiform area of the node of Tawara; NT, fasciculus at the level of the bundle of His, FH, and of its left branch, BGH. OD, right auricle; TR, tricuspid valve; VG, left ventricle.

Autopsy. July 12. On opening the thorax the pleural cavity was found to contain several cubic centimeters of a lemon yellow fluid. The lungs showed edema, with emphysema of the anterior borders. The liver weighed 1650 grams, was quite pale, and on section the surface was slightly wavy but without sclerosis. The gall-bladder contained some light colored bile. The spleen weighed 310 grams and was quite firm in texture. The kidneys weighed together 270 grams. The cortex was slightly thinner than normal, the fatty capsule thicker. The surface was somewhat irregular and showed a small brownish yellow infarct.

Heart: Much blood escaped when the right pulmonary hilus was incised. Nothing abnormal was found in the several orifices with their valves.

Histological Examination of the Ventriculonector: A block was removed from the septum. It was limited above by an incision passing above the orifice of the coronary venous sinus, at the point of junction of the anterior and septal segments of the tricuspid valve; below by a parallel incision 1 cm. below the first; and by an incision perpendicular to the preceding, passing posteriorly through the orifice of the coronary venous sinus, and anteriorly by an incision parallel to the foregoing, and 4.5 cm. distant. This block was embedded in paraffin and cut into serial sections, each 7 microns in thickness, whereby 1200 consecutive sections were obtained. One section in five was taken from the first 700, then one in ten from the remainder. They were numbered from above downward, the heart being supposed vertical with the apex below.

The sections were stained with hematein (van Gieson), fuchsin (Weigert), the latter being selective for elastic tissue, and Mallory's mixture, which stains muscular tissue red and connective tissue blue.

The first trace of the node of Tawara appeared at about section 1040. At section 930 the node was already well developed and centered by its artery; and at section 645 the node could be seen centered by its artery, and also, on the other side of the interventricular septum, the left branch of the bundle of His, lying under the endocardium of the left ventricle.

At section 520 the bundle of His appeared continuing the node of Tawara and soon entering the fibrous septum, at 485 it had passed completely through the septum, and at 455 and 434 the continuity between the node of Tawara, the bundle of His, and its left branch was seen clearly, the whole forming one and the same structure, which measured 12 mm. on the section. Toward 225 the node of Tawara disappeared and the bundle of His alone was evident. The right branch of the bundle of His, seated entirely in the fibrous septum, extending a distance of 6 mm., appeared now for the first time. Finally at section 155, there remained nothing more than a small portion (about 2 mm.) of the bundle of His in the fibrous septum. All of these measurements should be increased by about one-third to allow for the actual dimensions in a piece of fresh, non-dehydrated tissue.

The node of Tawara and the bundle of His with the initial portion of its branches were examined in sections 35 microns apart. The interval (1/30 mm.) was too slight for any lesion to escape detection. The major portion of the whole structure, which I regard as a single tract called the ventriculonector, was found to be absolutely free from any lesion. I had already shown the absence of any lesion in the bundle of His in a case of Stokes-Adams syndrome, reported in 1913.¹⁰ But I had not at that time examined the coronary system in detail, and the anatomical specimen, unfortunately mislaid during the War, has never been restudied.

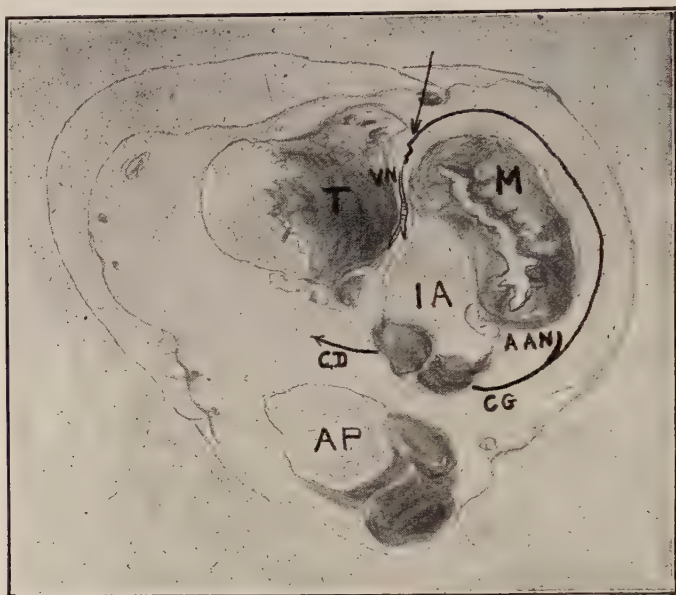


FIG. 180. The arterial channel between the aorta and the ventriculonector. Horizontal section of the heart (schematic). 1A, aortic infundibulum or chamber of expulsion of the left ventricle; CG, left coronary artery which gives off the artery of the ventriculonector; VN, the ventriculonector; CD, right coronary artery; AAN, artery of the atrionector; T, leaflet of the tricuspid valve; M, mitral leaflet. The arrow points to the seat of the stenosis.

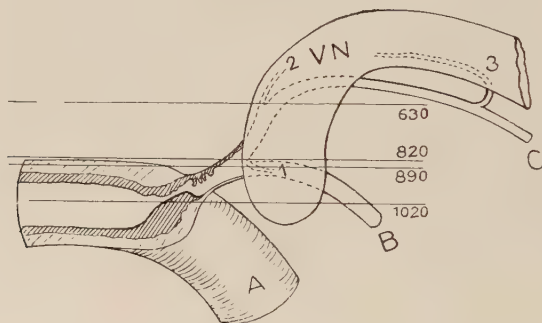


FIG. 181. Semischematic reconstruction of the arterial supply to the region of the ventriculonector. VN, ventriculonector; A, left coronary artery; B, artery of the septum; C, artery of the ventriculonector; 1, 2, 3, the special branches supplying the ventriculonector. The localization of the arteritis and the section levels 630, 820, 890, and 1020 are indicated.

Study of the Arterial Circulation of the Ventriculonector. It is known that the artery which supplies the ventriculonector rises on an average 92 times out of 100 from the right coronary and 8 times from the left coronary. We therefore followed the right coronary up to its aortic origin, but dissection showed us that this vessel did not reach as far as the posterior interventricular groove, and so did not give off the posterior septal arteries.

We then followed the left coronary and by dissection uncovered it as far as the posterior interventricular groove. At this point it entered the block removed for examination of the bundle of His, and which had been cut into 1200 serial sections (fig. 180). In its course from the aortic origin to its entrance into the block, this vessel gave off the anterior interventricular artery and the artery of the atrionector.

The study of the serial sections enabled us to reconstruct the route by which the arterial blood supply reached the ventriculonector. Figure 181 represents this reconstruction. We see that the left coronary (A) where it curves downward to become the posterior interventricular artery, gives off a considerable branch which passes from before backward and, after curving with convexity upward, becomes the artery of the septum. This artery is marked B in all our pictures. It is from this artery (B) that the artery of the ventriculonector (C) which is another septal artery, is given off. But although the branches which it sends to the ventriculonector, even if they are not the most important as regards size, have nevertheless such a definite functional value that it has seemed to me legitimate to give to the artery from which they arise a name indicating the important area which they supply.

It is always a matter of difficulty to give a sufficiently explicit name to an artery, and the present case is an instance. We see in the illustration the branches destined for the ventriculonector. But that marked 1 is given off from B, below the point where the artery C is given off, while the two other arteries, 2 and 3, (the latter having a retrograde course) are given off from artery C.

The route followed by the blood going from the aorta to the ventriculonector was, successively, artery A, artery B, and artery C. Along this route there were evidences of endarteritis, specially pronounced in A, immediately above the origin of B. The artery B also showed certain plaques quite apparent in figures 182 and 183; and in branch 3, given off from artery C, there was again a narrowing of the lumen.

But all these lesions, however marked they were had only a minimal importance, for they were most developed in a wide artery, the coronary A, and the amount of arterial blood carried to the ventriculonector was probably only slightly diminished by this. There would be nothing surprising then if we found similar lesions in the hearts of subjects who had never throughout life, so far as known, presented any disturbance of ventricular rhythm.

But at the point where B is given off from A the endarteritic nodules, those in A and those in B (figure 183) are opposite each other and together they form a sort of plug blocking the orifice of B. The importance of the lesion is due here more to its localization than to its size. The contraction of the muscular layer of the coronary, A, probably contributes when it is very marked, to the augmentation of the obstruction of B, by forcing the plug more closely against its point of origin from A. In artery B, by the side of the plaques of proliferating arteritis, there will be seen a zone of arteritis where the predominant element is a notable rarefaction of the muscular tunic. This alteration is found over a length of about 3 mm. The tunic is reconstructed below, as is shown by a section of the same artery, B, within the septum (fig. 182).

Absence of Lesion of the Ventriculonector; Stenosing Lesion of the Arterial Supply of the Ventriculonector—These two findings which sum up the description of the case form of it a document of the same value as the protocol of a physiological experiment.¹¹

Case II. Felix F., aged 67, chimney builder; entered the medical service of the Hôpital Cochin, under Prof. Vidal, June 4, 1925, for asystole. There was no history of infectious disease; no evidence of syphilis; the Bordet-Wassermann and Hecht tests were negative.



FIG. 182. The arterial channel at the level of the stenosis. Horizontal section of the septum between the two chambers of the heart. OD, right auricle; SC, venous coronary sinus; OG, left auricle; VG, left ventricle; M, mitral valve; S, interventricular septum; A, left coronary artery; B, branch of the coronary from which arises the artery of the ventriculonector, properly speaking; NT, Towara's node. (Section 1022.)

In 1922 he underwent prostatectomy, and returned at the beginning of 1925 to the same surgical service for hydrocele of the tunica vaginalis. It was some time after his second stay in the hospital that the first symptoms of cardiac insufficiency appeared. The patient first noted an abnormal fatigue with a little edema about the malleoli. Toward April 15 the symptoms became aggravated; there was effort dyspnea, then soon afterwards decubitus dyspnea with crises of asthmatic type. By the end of May there was a considerable diminution in the excretion of urine.

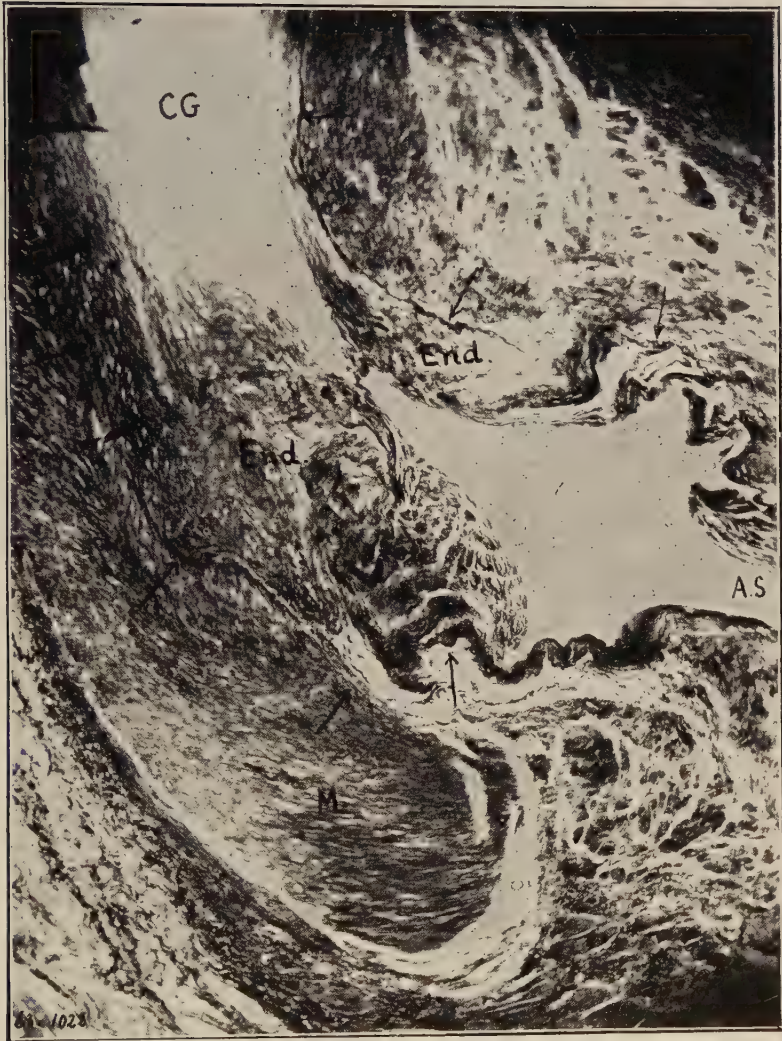


FIG. 183. The arterial lesion. Enlargement of the portion enclosed in the quadrangle in the preceding figure. CG, left coronary artery (A in figure 182); AS, artery of the septum (B) which gives off the artery of the ventriculonector (C); M, myocardial artery, End., endarteritis narrowing the lumen of the coronary artery and tending to form a plug before the orifice of the artery AS.

When the patient entered the surgical service for the third time it was soon found that the oliguria of which he complained was not due to any local cause, such as hypertrophy of the prostate, but was caused by cardiac insufficiency. Medicinal treatment was instituted, but through an error in the interpretation of a prescription, the patient, in place of taking 50 drops of a 1:1000 solution of digitaline, distributed over 6 days, was given the total quantity on each one of the six days, equal to 6 milligrams of digitaline. The only abnormal phenomenon resulting from this was a marked slowing of the pulse, this being due, however, less to an exaggeration of the ventricular bradyrhythmia than to a bigeminism from extrasystole. The situation remaining unmodified the patient was transferred to the medical service. On admission he presented a complete picture of total cardiac insufficiency; intense dyspnea, cyanosis of the face with distention of the jugulars, anasarca, and effusion into the peritoneal and pleural cavities, especially on the right side (the fluid contained large endothelial cells, lymphocytes, and polynuclears). The liver was tender on pressure and could be felt four finger breadths below the costal arch; there was a hepato-jugular reflex. Marked oliguria (300 cc. in 24 hours) was present, and albuminuria (1 gram per liter).

Examination of the heart revealed a notable enlargement of the cardiac area, the apex was displaced laterally, the right border extended considerably beyond the sternum, and there was an increase in the area of aortic dulness. There was a systolic murmur with maximum at the right second intercostal space and propagation into the vessels of the neck; no diastolic murmur. But the most remarkable sign was a quite constant bradysphygmia, between 32 and 36 during his stay at the clinic. No cerebral trouble, lipothymia, convulsive attacks, or syncope had attracted attention, and even the bradysphygmia had not been discovered. It is difficult to say when this may have begun. Nevertheless it had probably not been present in 1922, before the prostatectomy, for it is hardly likely that such a marked slowing of the pulse (one beat in every two seconds) would not have been detected at the time the blood pressure was taken. Also there was no mention of any abnormality of the pulse in the protocol recorded at the beginning of 1925, when the patient was again in the hospital on the surgical service.

The bradysphygmia was not modified by compression of the eyeballs. Despite its remarkable constancy, the pulse on several occasions dropped to 24. But this extreme slowness of pulse was not the expression of a correlative ventricular bradyrhythmia but of a bigeminism due to left extrasystole, this bigeminism being of the digitalis type (see fig. 184). The blood pressure, as recorded by the sphygmomanometer of Vaquez and Laubry, was 21.7 cm.

Treatment. The patient was put on a milk diet and kept in bed. The pleura was punctured three times, first on the right side, on June 11, 800 cc. being withdrawn; a second time on the right side, June 18, 850 cc. being withdrawn; and a third time on the left side, evacuating but 150 cc. This was followed by an improvement in the patient's condition. The edema diminished, dyspnea ceased, the ascites was absorbed, and diuresis was reestablished to the amount of about 2 liters a day. When this fell to 500 cc. a medium dose of theobromine brought it back to the usual amount. On June 23 the bigeminism disappeared; on June 29 the blood pressure was down to 13.6 cm.

On June 29 the bigeminism reappeared, the pulse being 24, but the patient felt better. Urine, 3.5 liters. Everything indicated a notable improvement, but suddenly at 2:30 in the afternoon the patient expired in his bed without a cry or convulsion. Five minutes before he had been talking to a patient in the next bed.

Autopsy was not performed until July 2d, but it had been possible to remove the heart

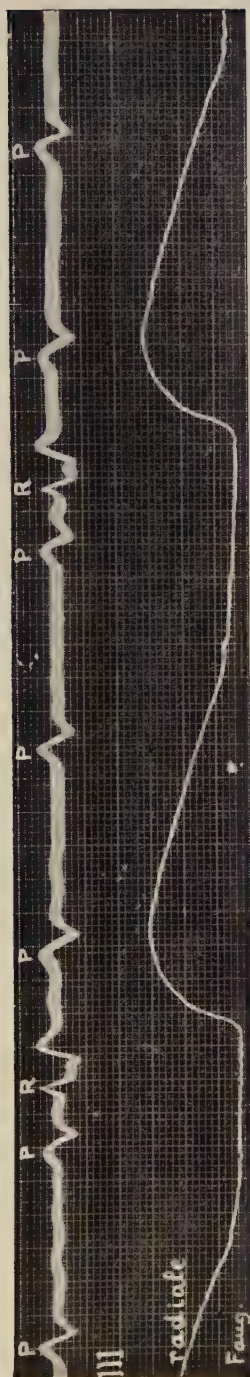


FIG. 184. Permanent ventricular bradyrhythmia. The ratio of the rhythms is $99:33 = 3:1 = 3$

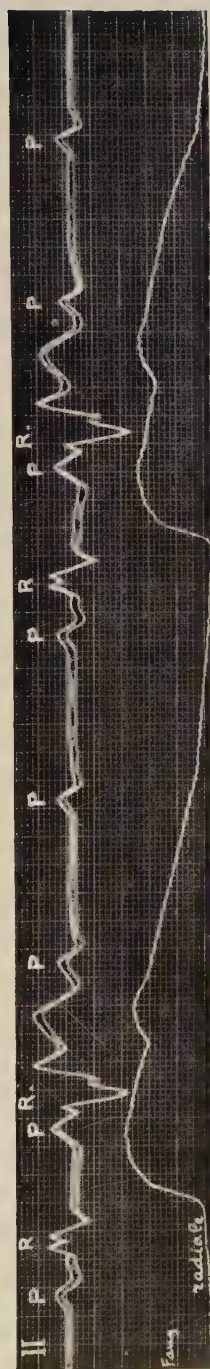


FIG. 185. Permanent ventricular bradyrhythmia. Allorhythmia from bigeminism of the ventricular series. In figures 184 and 185 the tracings have been retouched with water color, being too pale to admit of reproduction.

with but little delay, permitting a good fixation of the tissues. Examination of the organs revealed nothing worthy of report. The medulla oblongata was given to Professor Foix who had expressed a strong desire to make the examination. He reported as follows: "Macroscopically, in the different sections only cadaveric alterations were noted; microscopically, beyond the cadaveric lesions mentioned, there was no modification of the nucleus of the pneumogastric, save an increase of the pigment cells."

Macroscopic examination of the heart revealed nothing notable in the right cavities and pulmonary artery. On the left side the mitral ring presented a thickening of the free border, a little more marked to the left than to the right, and there were some yellowish streaks of sclerosis on the larger segment. The necrosis of the left ventricular wall, which was found on subsequent microscopical examination, passed unobserved, concealed as it was beneath the mitral ring. The aortic segments were thickened and their free borders presented facets where they were opposed to each other.

Study of the Electrocardiograms. On June 15 electrocardiograms were registered in the three leads at the same time as the radial pulse by Pachon's oscillogram.

The P waves follow regularly at the rate of 99, the ventricular complexes at the rate of 33 per minute. The ratio of the two rhythms is therefore a simple one of 3 to 1, whence the apparent constancy of the interval PR, measuring from summit to summit 18/100 second. The form of the ventricular complex is altered (fig. 184).

The tracing in figure 185 shows bigeminism by "left extrasystole," following at a brief interval after the regular complex. The tracing of the oscillogram shows an elevation with a double summit, the second attaining practically the height of the first,—a double ventricular contraction.

On another tracing the same rate is shown for the P waves but the rate of the ventricular complex is slowed, being no more than 24 to the minute. The ratio remains simple but it is now 4 to 1. This change took place in the short interval necessary to standardize the string in passing to a new lead.

Examination of the Ventriculonector: A block removed according to the technique previously described was cut into a ribbon comprising 1240 sections, each 7 microns in thickness. These sections were numbered from below upward, the heart being supposed to be vertical, with the apex below. One section in five was mounted from 1 to 500, 1 in 10 from 500 to 560, 1 in 20 from 560 to 1240. Most of the sections were stained by Van Gieson-Weigert, certain of them by the Mallory solution.

In the first section the tapering extremity of the node of Tawara was visible, the node coming to its full development in the succeeding sections. The bundle of His began to enter the septum at section 165; at section 290 it was clearly intraseptal; at section 340 the left branch, well differentiated, appeared to prolong the bundle (see fig. 186); it could be followed to section 385, when the bundle seemed to diminish in extent and the only portion still in view was a part of the superior convexity which diminished constantly in length. It was still perceptible at section 500, but had disappeared at section 520. Followed from section 1 to section 520 at intervals too close (every 30 microns) for any lesion, however minute, to pass unobserved, the ventriculonector (node of Tawara, bundle of His, and its branches) did not present any alteration, its continuity being nowhere interrupted.

This first finding was important for it brought a new proof of the possible absence of a lesion of the ventriculonector in an obvious case of discordance of auricular and ventricular rhythms. It is then proper in the present case, as in the first one cited, to study the arterial circulation of the ventriculonector.

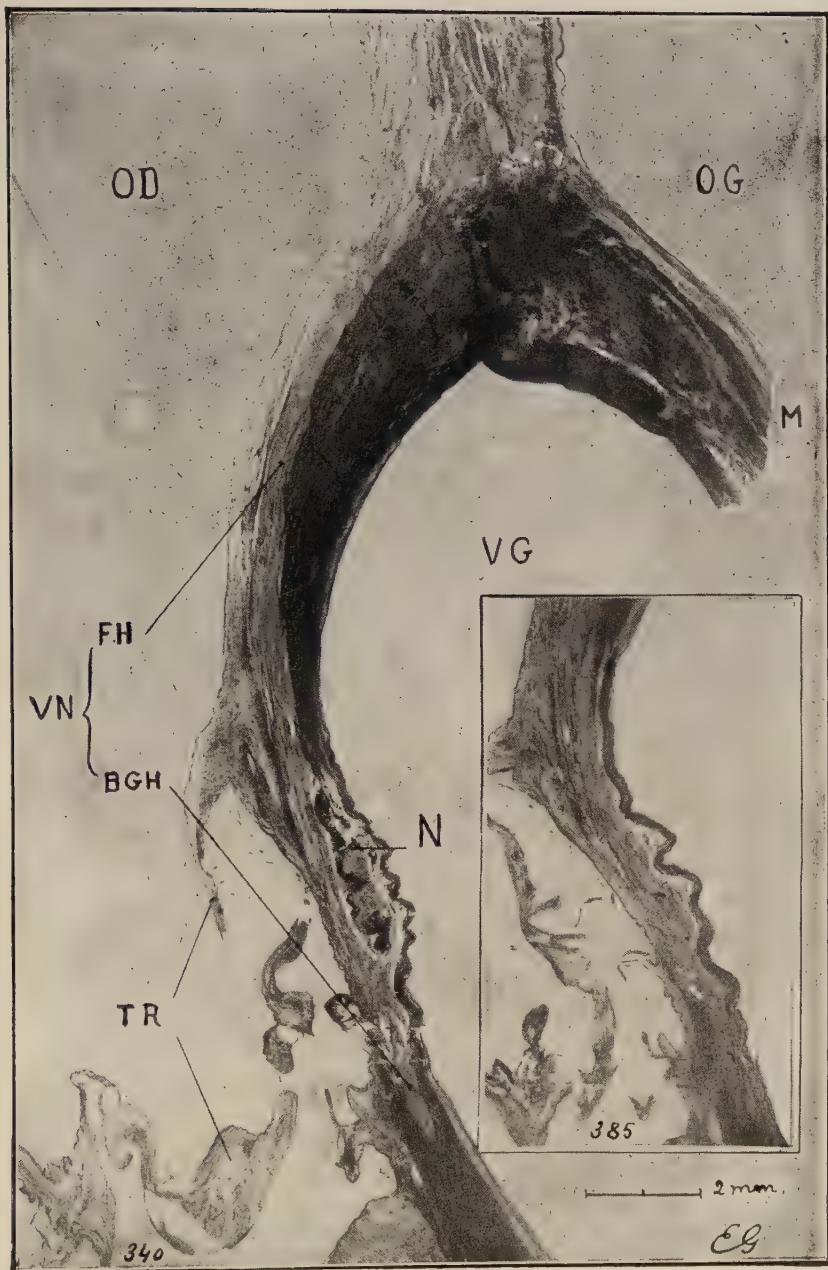


FIG. 186. The entire ventriculonector. Microphotograph of section 340 showing the details of the ventriculonector, VN, consisting in this section of the bundle of His, FH, and the initial portion of the left branch, BGH. This section was selected in order to show also the seat of necrosis of the fibrous septum, N, which seems to interrupt the course of the fibres of the bundle of His on its left half. These fibres are not interrupted but are raised up by the focus of necrosis. They are found restored through the whole width of the bundle of His in the microphotograph of section 385 shown in the rectangle. OD, right auricle; TR, tricuspid valve; OG, left auricle; VG, left ventricle; M, mitral valve.

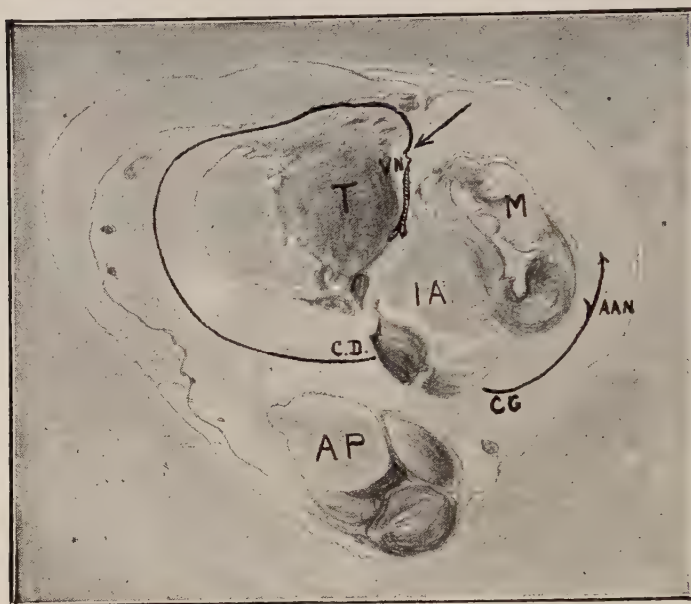


FIG. 187. Arterial channel between the aorta and the ventriculonector. Horizontal section of the heart (schematic). IA, aortic infundibulum, or expulsive chamber of the left ventricle; CD, right coronary artery which gives off the artery of the ventriculonector, VN; CG, left coronary artery which gives off the artery of the atrionector, AAN; T, tricuspid leaflet, M, mitral leaflet. The arrow points to the seat of the stenosis.

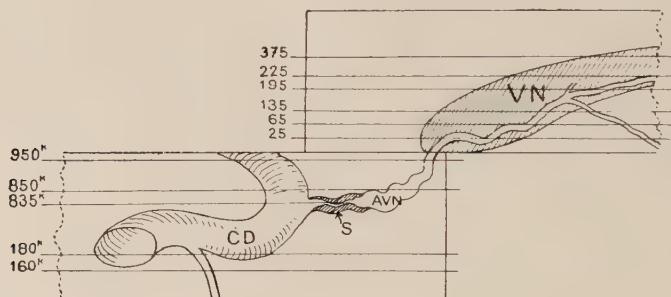


FIG. 188. Semischematic reconstruction of the arterial channel at the side of the ventriculonector. VN, ventriculonector; CD, right coronary artery; AVN, artery of the ventriculonector. The arteritic localization, S, and some of the section levels in the two blocks are indicated. Only one part of the block is here represented.

Arterial Circulation in the Ventriculonector: Dissection of the coronaries enabled us to determine clearly the origin of the artery of the ventriculonector. It was given off from the right coronary (fig. 167). The left coronary gave off the artery of the atrionector.

We then took a second piece of the septum below the one containing the ventriculonector, continuous with the latter posteriorly and extending to the posterior border of the heart.

This block was cut, like the first, into a ribbon of 1000 sections of 7 microns each in thickness. They were also numbered from below upward, the numerals being followed by an asterisk to distinguish them from the sections of the first piece. One section was taken in every 20 from 1* to 200*; one in every 5 from 200* to 410*; one in 20 from 410* to 740*; one in 5 from 740* to 960*; and one in 20 from 960* to 1000*. At certain points of special interest all of the sections were taken. All in all, from this piece, as from the first, 278 sections were stained and examined.

In section 180* the right wall of the right coronary artery appeared in the block, being lodged here in the adipose tissue of the groove which separates the right auricle from the left ventricle. The coronary was readily followed, section by section. In section 740* the artery of the ventriculonector was given off and this was readily followed to the last section 1000*. Here we passed from the second or inferior block to the one above where we had already located the bundle of His. In the first section the artery of the ventriculonector was again picked up. Figure 188 shows a semi-schematic reconstruction of the course of the right coronary artery and of the artery of the ventriculonector in the two superposed blocks.

The artery of the ventriculonector, immediately after its origin from the coronary artery, showed for about 2 mm. inflammatory lesions of the chronic type, considerably narrowing its lumen. All of the sections between 835* and 850* were stained and examined one by one. These included the greater part of the stenosis, of which we wished to obtain a precise idea. It was at section 840* that the lumen was the widest. The stenosis was considerable as is shown in the accompanying microphotographs (figs. 189 and 190). The result of it was that the supply by the artery irrigating the ventriculonector was *greatly reduced*.

The finding is of the same order as that of the preceding case, showing the presence of a plug of endarteritis at the origin of the ventriculonector artery from the coronary. We may add that beyond this stenosis there was another narrowing which was less constricted. The artery further along was tortuous, and considerable differences of calibre gave evidence of an alteration of its walls. Examination of the sections of the two blocks showed two other interesting lesions, one in the fibrous septum, the other, in the second block, in the wall of the left ventricle.

The lesion of the fibrous septum was found in the sections from 270 to 385. The microphotograph of section 340 gives a good idea of it (fig. 186). It may be seen that one part of the fibrous septum is fragmented and poorly stained and this alteration measures 2 to 3 cm. in length, 1 mm. in breadth, and about the same in depth. The fibrous tissue here is either necrosed or on the way to necrosis, as shown by the metachromasia. In the Mallory stained sections the color is red instead of blue, while in the sections stained with Van Gieson the color is a much deeper red than that of the connective fibres which remain sound.

In the lesion and its confines there was no trace of an inflammatory process, neither accumulation of young connective tissue cells nor foci of diapedesis. There existed, without question, a local necrosis from lack of arterial irrigation. This focus of necrosis of



FIG. 189. The arterial channel at the level of the stenosis. Horizontal section of the septum between the two hearts. Between the right auricular wall, PAD, and the left ventricular wall, PVG, in the adipose tissue bounded posteriorly by the posterior border of the heart, BP, are located the posterior interventricular vein, VIP, cut a little above its opening into the coronary sinus, and the right coronary artery, CD, from which arises the artery of the ventriculonector, AVN. In the rectangle, S, is seen the stenosis of this artery; N, necrosis of the left ventricular wall. Section 843*.

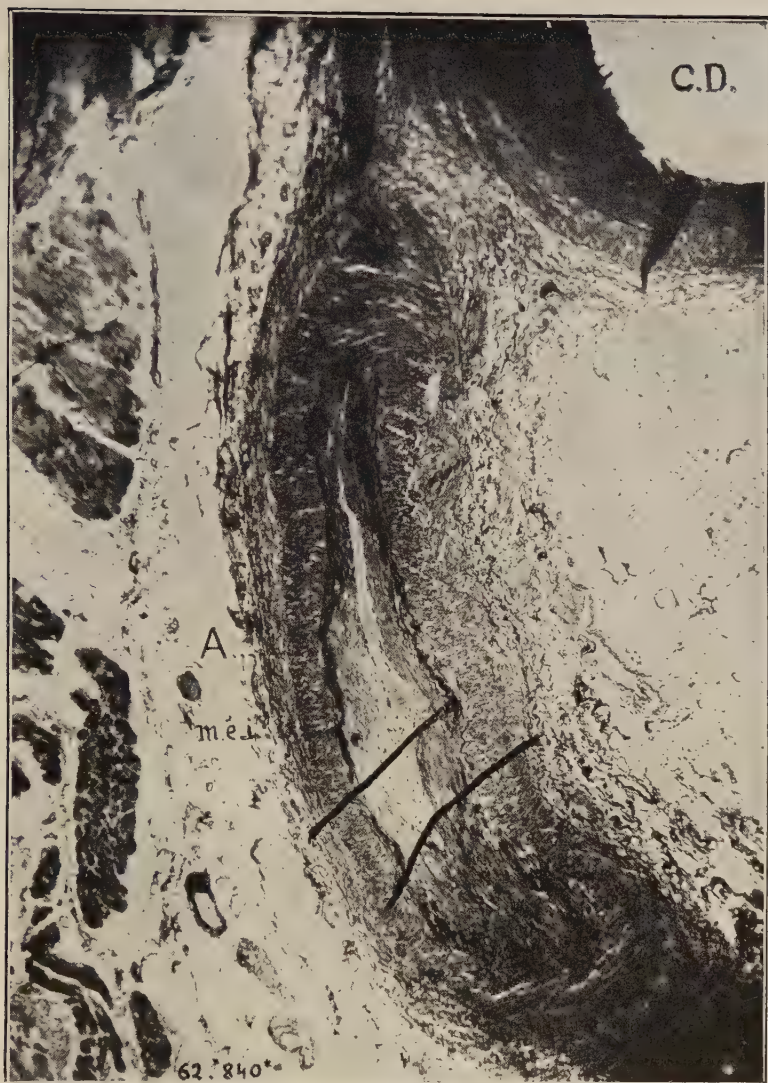


FIG. 190. The arterial lesion. Enlargement of a part of the preparation 840* corresponding nearly to the thickness of three sections, *i.e.*, $21\ \mu$, in the portion enclosed in the quadrangle in the preceding illustration. CD, right coronary artery. The artery of the ventriculonector A is sectioned longitudinally. The stenosing endarteritis is clearly shown, for the internal limiting elastic membrane, *m.e.*, stained by Weigert, separates the muscular coat from all the newly formed endarteritic tissue to its inner side. The normal lumen of the vessel corresponds to the black line of the elastic membrane. This section was chosen because it is at this level that the lumen of the vessel was widest; at each side of this the stenosis was much more marked.

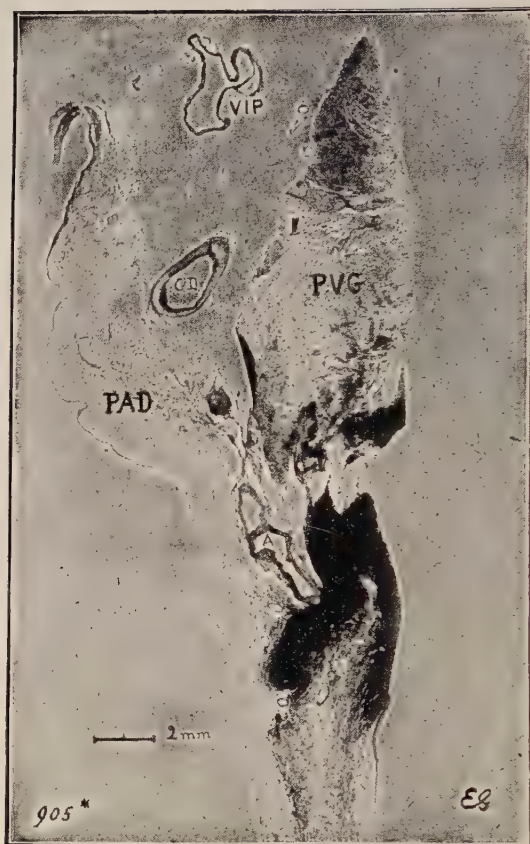


FIG. 191. The necrotic lesion of the left ventricular wall. Horizontal section of the septum at the level of section 905*. The left ventricular wall is completely destroyed up to where the irregularly dilated and tortuous artery, A, of the ventriculonector crops out. On each side of the loss of substance, the necrosed and metachromatic ventricular wall was stained a wine red by van Gieson; it comes out black in the microphotograph.

FIG. 192. Permanent ventricular bradyrhythmia. The two parts of the tracing were taken at a few seconds interval without changing the lead. Note the inversion of the ventricular complexes, the interpretation of which is difficult. This tracing was taken after a trial of amyl nitrite. The ratio of the rhythms is 120:37.



FIG. 192

the fibrous septum was in relation below with the portion of the septum which immediately adjoined the interventricular septum.

Toward the left the necrotic focus did not extend as far as the endocardium of the left ventricle, a thin layer of the fibrous septum intervening. The only lesion evident in the latter was a slight wrinkling of its fibers. Toward the right and at its upper portion the necrotic focus had much more interesting relations with the bundle of His. The fibers of this bundle also contrasted by their normal appearance with the lesion of the fibrous septum, the transition from the living to the dead portion being abrupt. Anteriorly the necrotic portion appeared to rest in a cup formed by the bundle.

The appearance of the section might lead to an error of interpretation, for one would be tempted to think that there had occurred a partial mutilation of the bundle of His. It is probable that such an error has been made at times, and this has led to the conclusion that there was a lesion of the bundle, at least in part. But when the lesion was followed, section by section, it became evident that this appearance was deceptive. The bundle of His had been raised by the focus of necrosis, in about a third of its thickness toward the left. Very soon, in section 385, for example (insert in fig. 186), it reappeared in its entire width. In section 340 (fig. 186) the appearance of a cup holding the anterior extremity of the necrotic focus was due to an oblique section of the fibers dipping down from the superjacent planes. From this it is evident that the only conclusive examination is one made on a complete series of sections.

We could not find in our specimens the obliterated arteriole which supplied this region and which must have been of extremely small calibre, considering the limited area of the necrosed region. It would have been most satisfactory if this obliterated vessel had been found, but the labor involved in taking a third piece of tissue from the septum subjacent to the other two, and preparing and examining a new series of sections, seemed to us out of proportion to the importance of the result, for the cause of so typical a lesion could not be in doubt.

The lesion of the left wall of the ventricular septum was opposite the mitral ring and a little below the zone of insertion of the latter. It was very important, for it involved the entire left wall of the ventricular septum, extending to the adipose tissue which separated it from the right auricular wall of the septum. In height it began at section 750* and extended to section 1000* with a maximum at about section 940* represented in figure 191. It was found again in sections from the first block, sections 1 to 200. It extended for a distance of 1.5 cm. of the dehydrated tissue.

All of this region of the ventricular wall presented signs of necrosis with metachromasia and an amorphous appearance of the tissues, and in places actual loss of substance, incompletely filled with loose connective tissue. The loss of substance resulted in the formation of an infundibuliform cavity opening out of the left ventricle, with its apex buried in the fatty tissue interposed between the left ventricle and the right auricle. The metachromasia at the parts at the margin of the loss of substance was particularly distinct for by Van Gieson's mixture the normal myocardiac wall was stained yellow and the necrotic part a lively red. With the Mallory stain the normal part assumed a violaceous red color, the necrotic portion a clear red tint.

As in the fibrous septum, we have here to do with a necrosis of the left ventricular wall by obliteration of one of the septal arteries supplying this territory. We have not found the precise point of obliteration in our sections, and for the same reasons, as given above, we have not judged it profitable to push our researches further in this direction.

Here again there is a marked contrast between the necrotic region and the healthy

parts which lie to the right of it, namely, the adipose tissue and right auricular wall. The demarcation is abrupt, as of two independent vascular territories.

We would note that the right coronary artery, before giving off the posterior inter-ventricular branch, presented a slight degree of stenosis.¹²

Case III. Nicolas Z., aged 71, entered the Hôpital Cochin in the service of Dr. Gautier, January 10, 1927. He came from the Hôtel Dieu, where he had been for three months, suffering from chronic bronchitis.

Examination on admission confirmed the diagnosis given by the patient. There was a frequent cough with ordinary mucous expectoration. On auscultation of the lungs coarse, sibilant bronchi were heard with numerous subcrepitant râles disseminated throughout both lungs. Examination of the heart revealed the existence of an aortic insufficiency from aortitis characterized by a loud double murmur at the base. The blood pressure was elevated (23-10.5 by the Pachon oscillometer). The capillary pulse was very distinct. On admission it was remarked that there were occasional extrasystoles and the radial pulse was full and bounding, and beating slowly, about 40 to the minute. This rate was not increased by movements, for the patient could get out of bed and walk rapidly, returning to bed without having increased the rate of 38 noted before the test.

Electrocardiograms, taken by Dr. Benard, showed a complete discordance of the auricular P waves and the ventricular R waves. While the former succeeded one another at the rate of 84, the rate of the latter was 40. The ratio of the two beats was then $\frac{84}{40}$. Nitrite of amyl caused an acceleration of the auricular beat only. Before the test the ratio was $\frac{9}{7}$, and after the test it became $\frac{12}{7}$ (fig. 192).

It will be noted in the tracing that the ventricular complex is a little abnormal; the base of the initial event QRS is enlarged, measuring 10/100 second in place of 5/100 or 6/100. The peak R is notched at the beginning of the descending limb. Finally, I would point out that in one of the tracings the initial event QRS of the ventricular complex in Lead II, at first positive is later completely reversed and becomes negative. The terminal event T is also modified. It is diphasic in the two halves of the tracing taken at a few seconds interval. The first phase of the complex is always negative, the second positive; but in the second half of the tracing the positive phase is longer and the peak higher while the negative phase is shorter. This, however, is not exceptional; and the fact that the initial events of the ventricular complex may vary at this point in the same subject in the same Lead, and at the same relative moment, should render one circumspect in seeking to attribute a precise significance to such variations. In particular it is difficult to accept the opinion that events of positive type are characteristic of right preponderance, and those of negative type of left preponderance. It is wise to confess that we do not know what value should be attributed to them.

When questioned as to his previous history the patient stated that in his youth, between the ages of 15 and 20, he had had convulsive attacks with loss of consciousness. These disappeared, but from time to time he had syncopal attacks. Quite recently again he had had these fainting spells and in addition he had had numerous attacks of vertigo and these also had increased of late. He denied all syphilitic infection, and no evidence pointing toward this could be found, unless one would regard the aortitis present as of luetic origin, which seems to be hardly warranted. The tendon reflexes were normal; the pupils were equal and reacted well to light; there was no leucoplasia buccalis. There were no sensory disturbances discovered on a rather superficial examination. The Romberg sign was absent.

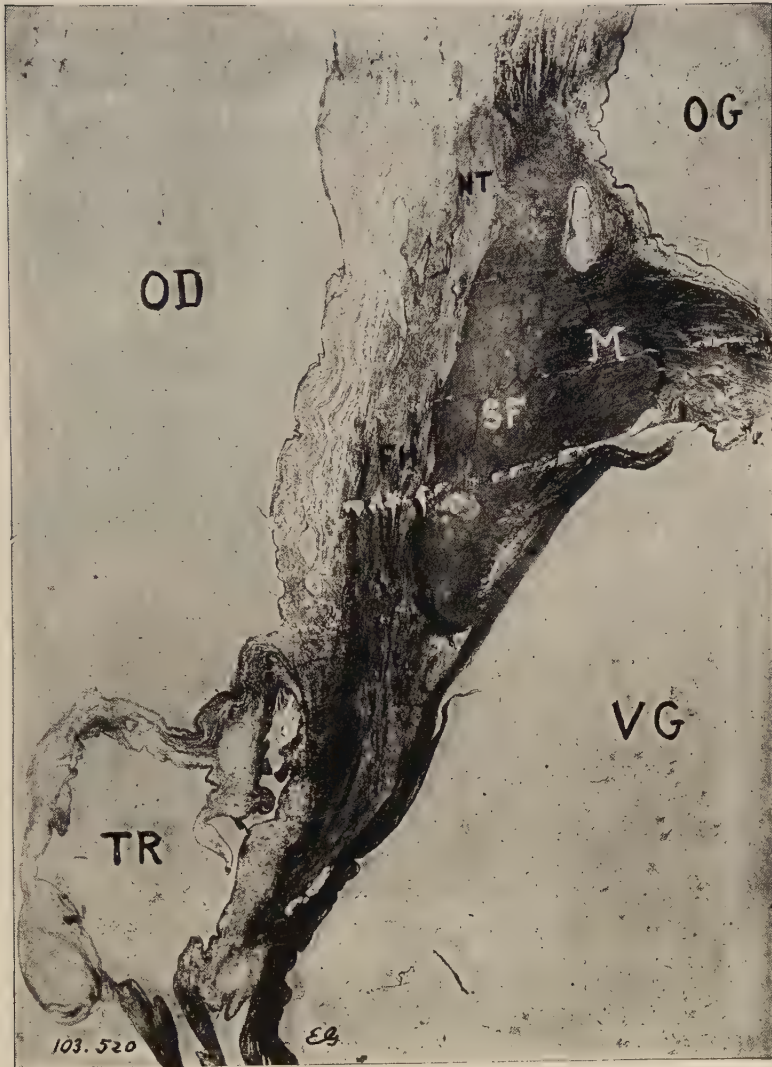


FIG. 193. The entire ventriculonector. Microphotograph of section 520. The ventriculonector, NT-FH, is lodged in the septum which at this level separates the right auricle, OD, from the left ventricle, VG. Tr, tricuspid valve; M, large flap of the mitral valve; OG, left auricle. The septum and the base of the septal implantation of the large flap of the mitral valve are thickened and contain mineral inclusions which nicked the razor and tore the section (van Gieson's hematein stain).

In the first days of March the patient complained of dyspnea and then had attacks of spasmodic coughing, the fits succeeding each other incessantly, interfering considerably with respiration. At the same time the expectoration became more abundant and slightly frothy. These crises of dyspnea occurred repeatedly and one night the attack was so severe that it was necessary to summon the interne. Next morning the patient seemed to have recovered and was free from dyspnea.

On the morning of March 14, when the orderly came on duty he found the patient inert, with face pale and slightly cyanotic, but without dyspnea. Following an injection

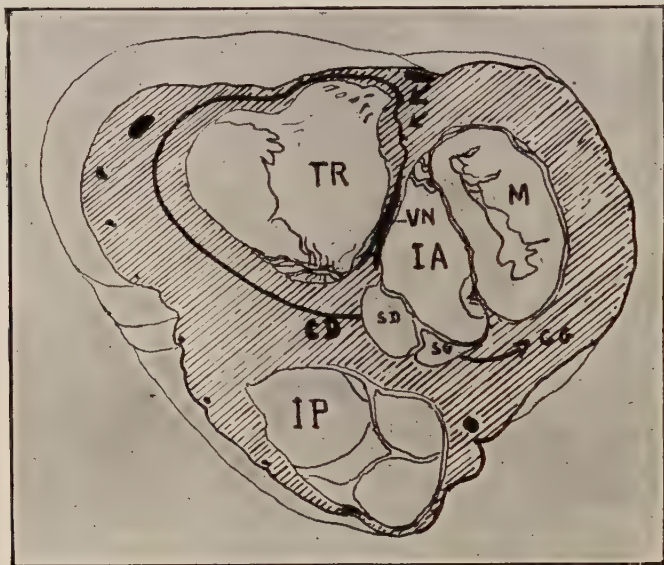


FIG. 194. The arterial channel between the aorta and the ventriculonector. Horizontal section of the heart (schematic). VN, ventriculonector; CD, right coronary artery giving off, at the level of the posterior border of the septum, the artery from which comes the branch to the ventriculonector. The two arrows point to the seat of the stenosis.

of camphorated oil and spartein he regained consciousness, and at the visiting hour he seemed normal; but he died suddenly in the afternoon of the same day.

Autopsy was performed March 16th. Bilateral serofibrinous effusion of the pleura, very abundant to the right was found, with old pleural adhesions. There were a few cubic centimeters of clear fluid in the pericardial sac. The kidneys were very large with somewhat adherent capsules. On section the surface was mottled and flocculent; the cortex was thin. The right suprarenal capsule was sclerosed; the left one was normal. The liver was sclerotic, creaking under the knife. The spleen was slightly enlarged. The aorta was enlarged but not sclerosed.

Examination of the Heart. The heart was quite large and globular in shape. The left ventricle was enlarged, but there was nothing abnormal in the right auricular and ven-

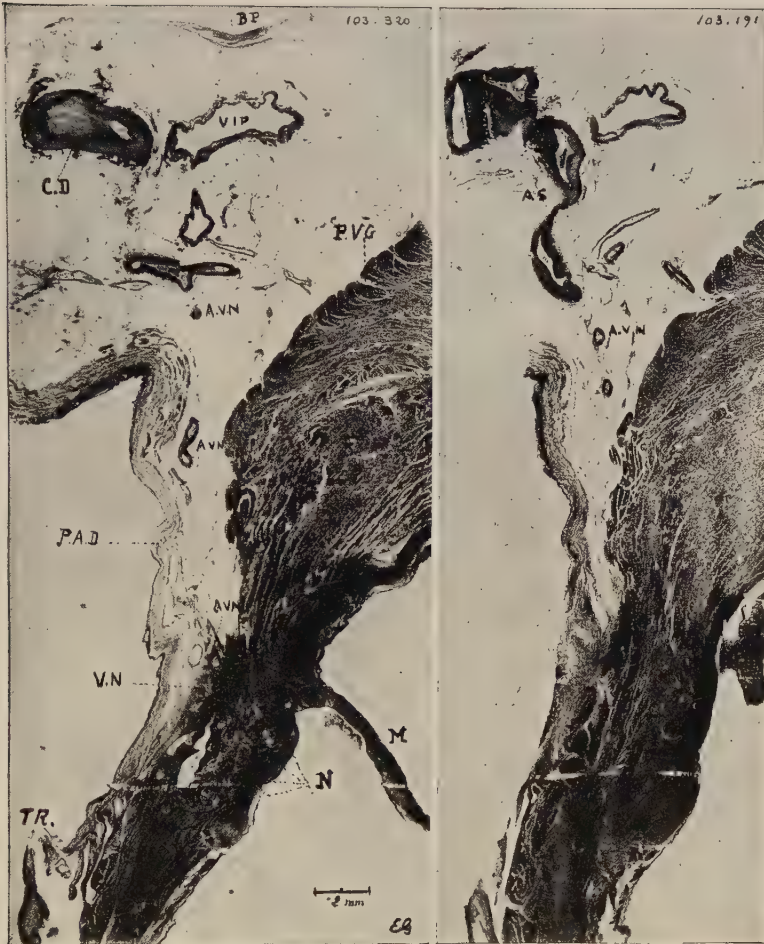


FIG. 195

FIG. 196

FIGS. 195 AND 196. The arterial channel at the level of the stenosis. Horizontal section of the posterior border of the heart and the septum at the level of section 320 (fig. 195) and section 191 (fig. 196). CD, right coronary artery obstructed by the endarteritis immediately above the origin of the septal artery, AS, which gives off the artery, properly speaking, of the ventriculonector, A. V. N., divided in several parts of its tortuous course; BP, posterior border of the heart at the level of the interventricular furrow; VIP, posterior interventricular vein near its point of discharge into the coronary sinus; PVG, wall of the left ventricle; PAD, wall of the right auricle; M, large flap of the mitral valve; Tr, tricuspid valve; VN, ventriculonector which begins to be seen in the sections at this point; N, foci of necrosis with deposits of mineral salts. The two sections here shown enable us to observe sufficiently the course of the septal artery, AS, and the artery of the ventriculonector, AVN, from its origin from the right coronary artery, CD, to its arrival at the ventriculonector, VN.

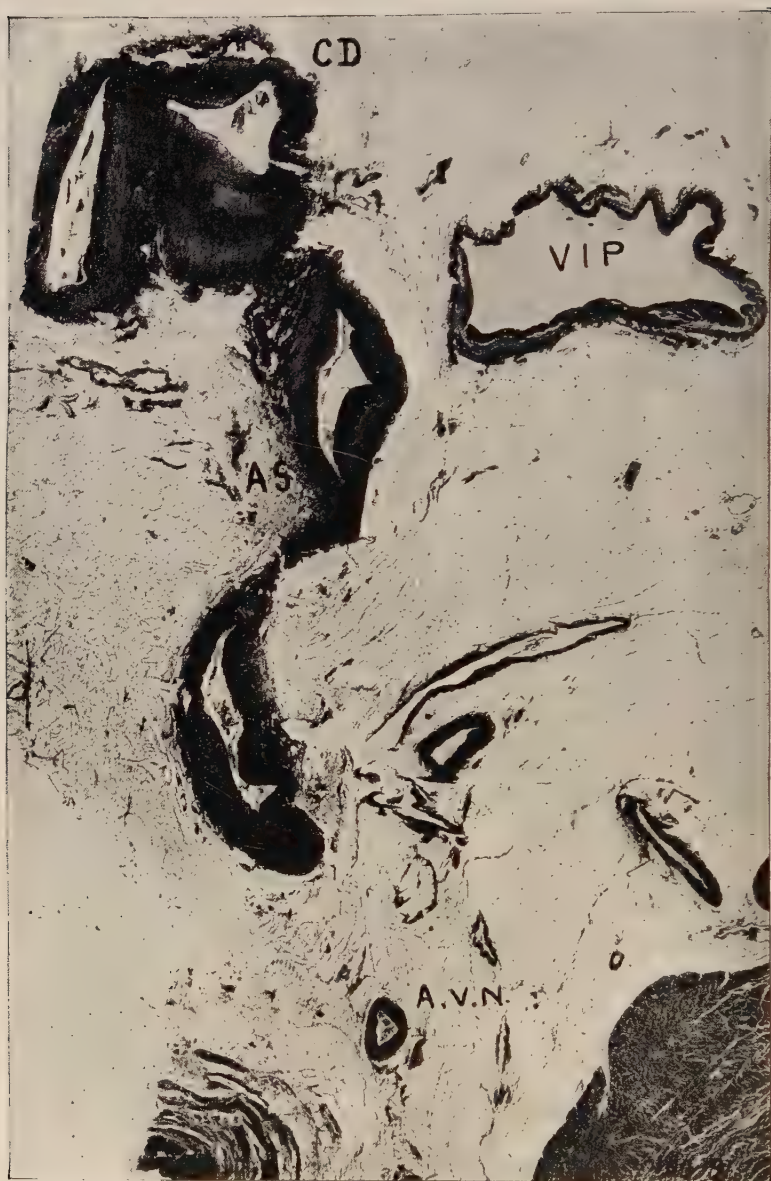


FIG. 197. The arterial lesion. Enlargement of the upper part of figure 196. The lettering is the same.

tricular cavities nor in the left auricle. The pulmonary segments were supple. The hypertrophied wall of the left ventricle measured 1.5 cm. at its thickest part. There was a slight thickening of the mitral ring, especially toward the auricular base and near its attachment to the septum. The aortic segments were thickened and the valve was incompetent. The right and left coronary arteries were thickened. The right coronary artery was very sinuous toward the posterior border of the heart, where it had been divided in several places; its wall appeared thick, irregular, and mottled with yellow spots; its external surface showed here and there small vessels, usually slightly or not at all visible on a normal coronary artery.

A fragment of heart was removed, following the usual technique and was cut into 1940 serial sections. One in every 20 was mounted and stained.

The ventriculonector began to come into view at section 220 and disappeared at section 880. It showed no lesion whatever. Figure 193 is a reproduction of the microphotograph of section 520, enlarged 12 diameters. We see here the ventriculonector and can follow its long axis for a distance of 52 mm., corresponding in actual length to a little more than half a centimeter.

The fibrous septum with which the ventriculonector came first into contact, and into which it later penetrated was thickened and certain parts were necrotic as shown by their staining a vinous red instead of the usual rose color, with Van Gieson. Finally, in numerous places there was a deposit of mineral salts, which resisted prolonged contact with an aqueous solution of hydrochloric acid, and notched the razor with which the sections were made, which accounted for tears in the same.

The artery of the ventriculonector in this heart arose from the right coronary (fig. 194) and at the point where the artery was given off from the coronary there was a very marked stenosis of the two vessels, of which the microphotographs (figs. 195, 196, 197) give a better idea than any description. The artery of the ventriculonector also presented in addition to this initial stenosis, numerous points of endarteritis which locally narrowed the lumen, but the maximum of the lesions was at the origin of the artery and in the coronary.

To sum up, the anatomical examination showed that the ventriculonector was intact and that the arterial supply was greatly diminished in consequence of a stenosis of the coronary and of the artery of the ventriculonector.¹³

I have been unable to collect more than the three preceding observations and these, thanks to the friendly collaboration of Dr. Giroux, in the first case; in the second, of Drs. Bénard and Hillemand, and in the third of Dr. R. Bénard, Dr. Cl. Gautier, and Mlle. Hermann.

The three cases cited above are in agreement. Clinically there was a bradyrhythmia of the ventricle. Anatomically the bundle of His was the seat of no lesion; but the arterial supply of the ventriculonector was markedly diminished by a stenosis of the artery of the ventriculonector or of the coronary from which the latter arose.

From this concordance established in the three cases, it seems legitimate to conclude with a maximum of probability, that it is an ischemia of the ventriculonector which determines the bradyrhythmia of the ventricle.

SIMPLE ANISORHYTHMIA

A certain number of the facts of anisorhythmia do not enter exactly into the types we have just described.

Neither of the two rhythms, auricular and ventricular, being excessively rapid or slow, we cannot regard either of the two as notably accelerated or slowed in relation to the other. Cases of this sort are rare, but very valuable from the doctrinal viewpoint. They demonstrate in a manner beyond dispute the independence of the rhythms, and in particular do they prove that if, strictly speaking, this independence may find a plausible explanation in the loss of conductivity of the bundle of His, it is inexact to hold that the idioventricular center which the "dissociated" ventricle obeys functions at a slow rate, for the ventricle beats at a rate which differs but little from that of the auricle.

The tracing in figure 198 shows us an auricular series which is quite regular, where the complexes follow each other at a rate of 55. In the ventricular series, becoming regular at the end of the tracing, the complexes follow each other at a rate between 55 and 60, that is to say, the rhythm is the same as that of the auricle. But it is not the same at the beginning of the tracing for the ventricular series is broken by coupled proiosystoles and the intervals between the complexes, whether deformed or not, are irregular and there is no fixed relation between the P waves and the R waves. After the ventricular series has become regular there is an appearance of grouping, but in each group P follows R, in place of preceding it.

The tracing in figure 199 shows us an appreciably regular auricular series where the complexes follow each other at the rate of 80. The ventricular series is also regular, the complexes following each other at a rate of 60, but there is no relation between the two series. Some minutes before, this subject had given a tracing of normal appearance in which there had been auriculoventricular isorhythmia at 60. It appears therefore that the momentary disturbance of the cardiac mechanism resulted from a puff of auricular tachyrhythmia which made the auricular count pass from 60 to 80, while the ventricular rhythm was not modified. I could just as well have placed this tracing in the chapter on discordant auricular tachyrhythmia, despite the but slightly more rapid auricular rate, and especially because this episode of relative tachyrhythmia was observed in a subject who presented, both previously and subsequently, crises of flutter.

The tracings in figures 88 and 89, which are shown at the end of the chapter on proiosystoles, catch the discordance of rhythm, alive, as it were. In figure 88 the auricular rate is regular, between 110 and 120, and the

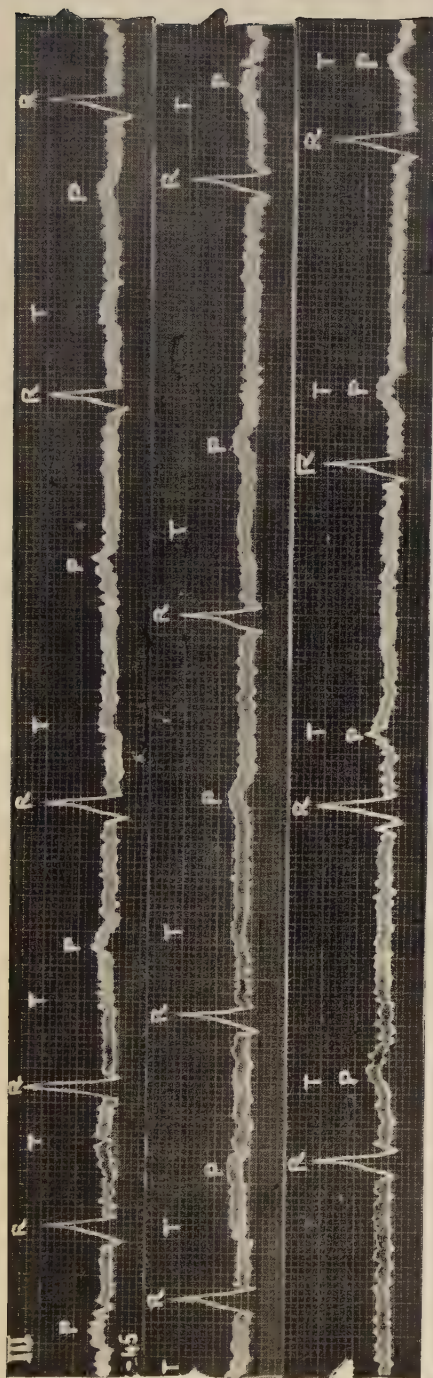


FIG. 198. Simple anisorhythmia. The auricular rate is practically constant at 55; the ventricular rate, quite irregular, becomes regular in the second half of the film (middle and lower tracings) and oscillates between 55 and 50. There is no regular grouping of the P and R waves.

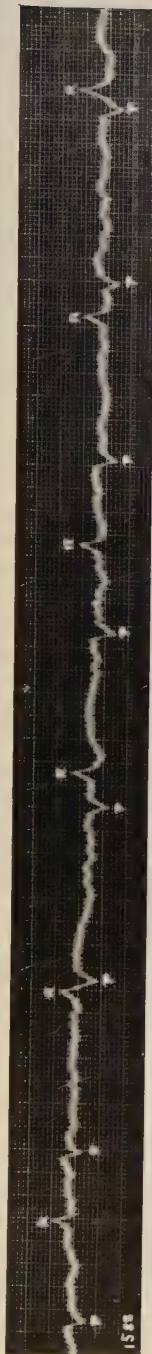


FIG. 199. Simple anisorhythmia. The ratio of the rhythms is $80:60 = 4:3$. The discordance of the auricular and ventricular series is evident. This patient subsequently gave typical tracings of anisorhythmia from discordant auricular tachyrrhythmia (flutter).

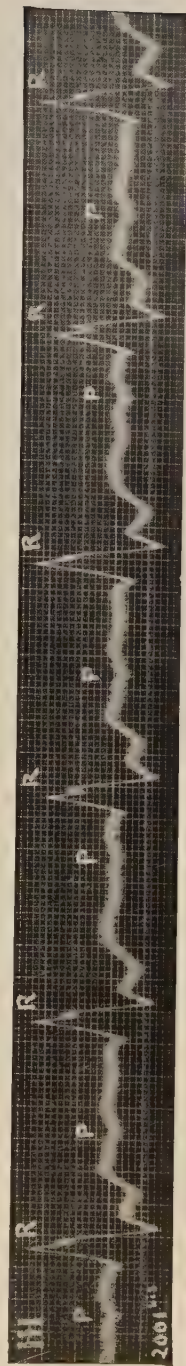


FIG. 200. Simple anisorhythmia. The discordance between the auricular and ventricular series is evident. Note the allorhythmia from bigeminism of the auricular series. This patient subsequently furnished typical tracings of anisorhythmia from discordant auricular tachyrrhythmia (flutter).

ventricular rate is the same, hardly broken on two occasions by the appearance of a proiosystole with deformed complex. On the whole, there seems to be concordance between the rhythms. But in the tracing shown in figure 89, taken immediately after that in figure 88, the two rhythms have changed. The auricular maintains its regularity, but falls to about 90 while the ventricular is broken regularly. A proiosystole with deformed complex, which is coupled with the preceding ventricular systole or normal complex, creates a special type of bigeminism, the couples succeeding each other at the rate of 35 per minute. There is no longer any apparent relation between the auricular and ventricular complexes and the discordance is evident.

The tracing in figure 200 is of interest, not only because of the anisorhythmia, but also because, while the ventricular series is regular at a rate of 84, the auricular series presents a very remarkable bigeminism. This tracing, like that in figure 199, belongs to a subject who had had for some days previously an auricular tachyrhythmia of flutter type.

If we compare all of these facts we shall see that they form part of the same continuous series characterized solely by the habitual discordance of the rhythms.¹⁴ This discordance may result from a relative tachyrhythmia or bradyrhythmia of one of the cardiac cavities; but it may also, in rare instances, constitute a simple anisorhythmia in which no one of the rhythms exceeds the other sufficiently to warrant our speaking of a relative tachyrhythmia or bradyrhythmia.

From the pathogenic point of view all of these facts belong to the same family. In all the same mechanism intervenes—a disturbance of equilibrium in the activities of the cardioneurons. So long as the equilibrium persists one has the impression that the auricular and ventricular series are intimately associated one with the other, that is to say that a P wave corresponds to an R wave; but let this equilibrium be broken, and the independence of the series, formerly masked, now becomes evident. It is no longer possible to preserve the illusion of a connection between P and R.

It is quite likely that different causes—humoral, toxic, or nervous—may influence the activity of the cardioneurons, and that these causes may act directly on the specific myocardium. But our belief is that it is through the medium of the circulation that these causes make their action felt most frequently. The disequilibrium of the rhythm is thus connected especially with the circulatory disequilibrium of the cardioneurons. There is a disturbance of nutrition sometimes of one of the cardioneurons, sometimes of both, and there result therefrom alterations in function of the auricle or of the ventricle or of both cavities jointly. All of these combinations have in fact been seen.

In general it appears that when the circulation of a cardionector is normal, the rhythm of the cavity controlled by it is also normal. But this normal circulatory régime depends on the ready entrance of arterial blood and exit of venous blood. The arterial supply may be increased or diminished, the result being active hyperemia or ischemia. The only change in the venous discharge can be in the way of obstruction, which causes passive hyperemia. With ischemia is associated bradyrhythmia; with hyperemia, tachyrhythmia. The result is therefore that disturbances of arterial origin may be tachyrhythmias or bradyrhythmias, while those of venous origin will be tachyrhythmias only. One might express in a rather homely, yet picturesque, way the relations between circulation and rhythm as follows: The cardionectors produce a normal rhythm when they are *pink*, a slow rhythm when they are *blanched*, and an accelerated rhythm when they are *red* or turn *blue*.

RÉSUMÉ

The classical theory of the cardiac mechanism has lost much of its simplicity by reason of the additions and embellishments which it has had to undergo in order to remain in harmony with facts successively discovered. Nevertheless, it always rests essentially on this idea, that the auricle is closely connected with the ventricle, whence the coördination of their movements.

We have observed that this coördination is lacking oftentimes: (1) Even in the normal state, where a certain play may exist between the auricle and ventricle; (2) still more when the heart is ailing, the independence of the auricular and ventricular rhythms being then at times so modified that we can no longer speak of coördination, however loose we may imagine it to be.

The auricle and ventricle, it is still said, are connected by a special bundle, the bundle of His. Now the bundle of His has the same structure as the sino-auricular node to which is attributed a very different function, namely, that of a motor center. Contrary to this opinion, we attribute the same function to these two similar formations which we call the cardionectors, the sinoauricular node, or atrionector, being to the auricle what the bundle of His, or ventriculonector, is to the ventricle.

Thus the atrionector and the auricle on the one hand, and the ventriculonector and the ventricle on the other, constitute two parts which act independently of each other. This conception is further confirmed by the distribution of the vessels of the heart. We have, in fact, shown that each cardionector has its own circulation, with one terminal artery for the atrionector and another for the ventriculonector.

It is therefore logical to suppose that a circulatory disturbance may affect only one of the cardioneotors, or both, although differently, as a result of which fact, the functioning of the auricle and that of the ventricle are seen not to be in partnership. So long as the circulatory equilibrium between the two cardioneotors is maintained, the rhythms of the auricle and ventricle remain equal. The independence of the two cavities is then masked and auricle and ventricle seem to be intimately associated with each other. This is the case with the normal heart. But when circulatory disturbance supervenes, however slight it may be, the equilibrium will be broken, and the auricle and ventricle will beat each at its own rate; their independence will thus be revealed and their assumed coördination will cease. The mechanism of the heart will become abnormal.

This is the conception which has seemed to us to accord best with the clinical data and the anatomical findings. It is then its own circulation which is the chief regulatory mechanism of the heart. The anomalies are due less to the condition of the heart itself, than to that of its blood vessels.

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- ¹¹ I presented a résumé of this case to the Academy of Medicine, Paris, session of December 29, 1925, in a communication illustrated by lantern slides. The complete history was published later in collaboration with Dr. R. Giroux, in the *Presse médicale*, February 27, 1926.

¹² I published this case in collaboration with Drs. René Bénard and P. Hillemand in the *Archives des mal. du Cœur*, May, 1926, under the title: "Un second cas de bradyrhythmie ventriculaire par sténose de l'artère du ventriculo-necteur."

¹³ I published this case in collaboration with Drs. René Bénard, Cl. Gautier, and Mlle. Hermann in the *Presse Médicale* of September 17, 1927, under the title: "Un troisième cas de bradyrhythmie ventriculaire par sténose de l'artère du ventriculo-necteur."

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Sans Tache

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